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EDITORIAL

NEW VISTAS IN ORTHOPÆDY

PATHOGNOMONIC lesions of bone, be they of constitutional or accidental origin, are as common in animals as they are in man and their ætiology and treatment follow similar patterns. It is in end results that the greatest divergence exists. In bone troubles, as in lesions in other parts of the body, one cannot expect that any form of treatment will always promote complete resolution of tissue and restoration of function.

In man the relief of pain and the restoration of some degree of functional activity is often accepted as a good result but in the animal world, no matter how exquisite be the local repair, if full functional activity is not restored the result is classified as a failure. Many men walk the streets and work quite cheerfully with skeletal deformities and abnormalities of gait which if they were exhibited by a quadruped would place the owner in peril at law.

The repair of injured bones is an important part of a veterinary surgeon's work. In the majority of cases the methods of treatment which have been practised for centuries serve well, but there are occasions when only open surgical interference can give one hope of a satisfactory result. Arbuthnot Lane was the great pioneer in this field in the last decade of the nineteenth century. He

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evolved an aseptic technique which revolutionised bone surgery. He introduced the internal splint. He was a master of his art but many who followed could not repeat his success. In the main this was due to two factors—a failure in the rigid aseptic technique and the electrolytic action of the metals used. These things have now been controlled, the first by the additional aid of sulpha drugs and the antibiotics and the latter by the development of non-electroyltic metal. These two factors have enabled human orthopædic surgeons to make great progress during recent years. Veterinary surgeons have not been slow to profit by this experience and in this issue we publish a short article describing a successful operation on a dog in which the damaged bone was replaced by metal.

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LIAISON BETWEEN TWO PROFESSIONS WITH SIMILAR INTERESTS*

By J. McCUNN, F.R.C.S., L.R.C.P., M.R.C.V.S.

London

I HAVE listened to and read many presidential orations when by custom and tradition the occupant of the chair has addressed those over whom he has been called to exercise some ephemeral authority. I have noted that it is customary and polite to display a modicum of pleasure on elevation to office and to express thanks for the honour which has been conferred upon one. As far as my researches go I have never heard or read a presidential address which expressed the opposite view so it may be that you are present on a unique occasion. The length and the portent of the speech would seem to be related to the egoism or erudition of the speaker and as the theme is not subject to challenge the audience must suffer in silence.

Although I have been a Fellow of the Royal Society of Medicine since 1923-24 and a member of this section since its inception I have never sought office, being quite content to be a back bencher, free to come and go as my interest and inclination willed. I have never professed to be academic or erudite and so when I was invited to fill the post I expressed some misgiving as to my fitness for the job and I communed within myself in search of a reason for your confidence. No doubt Sir Andrew Aguecheek had the same qualms.

As this section is one in which medical men and veterinary surgeons meet together to exchange thoughts and ideas in free and frank discussion, it may be it was thought that as I had a foot in each camp I would show bias neither one way nor the other and so be able to preserve the balance of power. This theory can be rejected for there has never been any rivalry or contest.

The men and women who are members of this section realise that the true art and science of medicine is indivisable whether it be applied to man, animals or plants.

I should like to think that the real reason for my preferment emanated from the knowledge that my interests have always inclined more to the clinical than to the purely academic side and that I might attempt to bring the clinicians, medical and veterinary, back to the forum which they seem to have avoided or neglected in recent years. I happen to be the editor of a veterinary periodical and I have noted in recent years that as the pens of the laboratory workers have reached a state of flux so, in direct proportion, those of the clinicians have registered a drought.

When this section was instituted way back in 1924, mainly by the initiative and enthusiasm of Sir Frederick Hobday and Sir Clifford Allbut, over 70 veterinary surgeons and a like number of medical men joined in the venture.

* Full report of Presidential Address delivered at a meeting of The Section of Comparative Medicine of The Royal Society of Medicine on October 19, 1955.

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Most of the members were drawn from the ranks of the clinicians who practised their profession in the field and were interested in the diagnosis and treatment of the diseases and abnormalities which confront a practitioner every day of his life. I expected to gain much from the meetings for there is a great extent of common ground between the practitioners of the medical arts and science.

This common ground has been recognised for a long time. The earliest physicians attended to man and animal and the cleavage occurred only when the doctrine, that man was the possessor of that divine attribute, a soul, was accepted. This placed animals on a lower plane and the ancient physicians gave them attention no longer for fear of debasing their profession. It took many centuries to regain the lost ground and the repercussions of the doctrine were felt in both fields and probably most harm was done to the forward progress of human medicine for, as man possessed a soul, his body was sacred and even dissection and investigation post-mortem were prohibited. The coming together of the two disciplines and the recognition again of a common ground was exemplified best in the late 18th and early 19th century when the veterinary profession as we know it to-day was going through the pangs of birth and mediæval conceptions and ideas were being cast aside.

We of the veterinary profession will ever be grateful to those medical men, M.R.C.S.s and L.S.A.s who joined our ranks and by virtue of their more specific training and scientific outlook, laid the foundations on which a learned profession could be built. I will mention some of them for we hold their names in great honour. Delabere Prichett Blaine, Moorcroft, Bracy Clark, William Percival and Joseph Goodwin. There were others who helped by allowing veterinary students access to their classes and by acting as examiners: E. Wilson, Abernethy, Hume, Travers, Cruikshank, Lister, Bell, Brodie, Bransley, Cooper, Jolly, Quain, Fordyce, Barley Babington and Bright.

Edward Coleman, the second Principal of the Veterinary College (1794-1839), was a surgeon but there is some debate, to which I do not entirely subscribe, as to whether his influence, during his long reign of nearly 50 years, was a good one. John Hunter, Henry Cline and Astley Cooper took an active part in the foundation of the Royal Veterinary College. John Hunter was a profound student of natural science. He practised in the human field but he could have won equal renown had he devoted his efforts entirely to veterinary science.

It is said that Moorcroft approached Hunter for advice as to whether he should take up veterinary science as a permanent calling. Hunter said: "If I were younger I would take up veterinary science forthwith." Here, also, is what Quain said in 1863: "He could not see why there should not be the greatest sympathy between the College of Veterinary Surgeons, the College of Physicians and the College of Surgeons. Their anatomy was very nearly the same, their physiologies were quite identical, their pathologies were identical and the practice of one harmonises very much with that of the other so that I do not see why there should not be the greatest sympathy between them."

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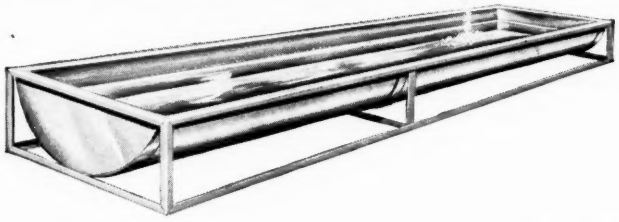

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Again on the same occasion Dr. Sharpey said: "Physiology is the same in both cases. Let me observe that we are largely indebted for the progress of physiology as a general science having no more special reference to man than to domestic animals, on which we so much depend, to members of the veterinary profession."

There can be no doubt that some of the medical men who joined the veterinary profession did so for purely altruistic motives, others did so because they recognised that their training and skill would command greater reward in the new field. Moorcroft, who established his veterinary practice at 224 Oxford Street, made a fortune in 16 years and then in 1808 went to India as veterinary surgeon and adviser to the East India Company at a salary of £3,000 per year.

The medical men who helped at the beginning were recruited mainly from the ranks of the surgeons. The physicians were very superior beings and they would not deign to pay attention to the diseases of the lower animals. On those occasions when they were asked by the government to give their views on epizootics such as cattle plague and foot-and-mouth disease they said that they would do so only from a sense of duty to the nation and apologised for debasing their profession. It would have been better for the nation if their sense of duty had not been so strong and they had remained silent. Their ignorance was absymal and especially so of the natural history of contagious disease. As some excuse one must remember that the ancient notion of the spontaneous origin of disease was not refuted completely until the latter part of the 19th century.

Professor J. B. Simonds, who was Principal of the Royal Veterinary College (1871-1881), veterinary surgeon to the Privy Council and adviser on veterinary matters to the Government, did not accept completely the contagion theory until 1875 when Professor Brown (later Sir George T. Brown), by experimental proof, was able to convince him of the contagious nature of pleuropneumonia in cattle. It is said that Simonds held that the liver fluke was of spontaneous origin and was the result of, and not the cause of, the liver disease with which it is associated.

It is strange that the theory of spontaneous origin was held so strongly and for so long even until late in the 19th century, by competent and eminent men of the medical and veterinary professions.

Primitive people in Africa had long recognised that pleuropneumonia of cattle was contagious. John Snow had traced a cholera outbreak to the Broad Street pump way back in 1795 and in the same year Thomas Peall had demonstrated at the Veterinary College that glanders could be transmitted from one animal to another. He took material from a farcy ulcer and smeared it on the nasal mucous membrane of an ass. The ass became glandered on the eighth day.

Undoubtedly the veterinary profession owed much to the medical profession in the 18th and early 19th centuries and when one considers the great number of surgeons and physicians who contributed freely to the literature and so added to the store of knowledge on matters appertaining to animal husbandry and disease and, in many cases, abandoned their human patients in order to devote their whole time to veterinary science, the integration between the two branches of medicine must have been very great at that time.

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Medical men played a great part in the founding of the Veterinary College and in its subsequent management. No doubt they recognised that the time had come when a systematic course of training, with requisite tests of efficiency, should be available for the better education of those who desired to follow a career in veterinary science. It is interesting to note that there was a properly constituted Veterinary College, in which all known facets of veterinary art and science could be studied, before there was a Medical School or College in London which could provide within its precincts similar facilities for a potential doctor.

At first the students resided at the Veterinary College and, according to the regulations then in force, they lived a spartan life, rising at 6 o'clock in the summer and 7 o'clock in the winter, and they were required to retire to their bedchamber at 10 p.m. They were not permitted to burn a candle in the night under the pretence of study. Lights were to be extinguished before the pupils went to bed and none was to remain up after 11 o'clock. The daily period of study concluded at 7 p.m. Prospective students had to submit evidence of a good education and be between the ages of 15 and 22. Preference was given to those who had some knowledge of surgery and pharmacy and the limitation of age did not extend to practitioners in medicine and surgery. The course was a comprehensive one, including zoology, botany, anatomy, physiology, pharmacy, pathology, shoeing and attendance in the infirmary and at operations.

It was considered that a term of three years was sufficient to complete the students' education provided that they were capable, assiduous and well inclined.

No doubt the medical men who were interested in the advancement of veterinary science had a hand in the designing of this curriculum, which was in many ways in advance of that followed by medical students in 1791.

I regret to say that this brilliant conception was brought to naught by a medical man.

The first Principal, Professor St. Bel, died in 1793 and a young surgeon, Edward Coleman, a protégé of Hunter and Cline, was appointed in his stead. He took office in 1794. Coleman, unlike the other medical men who had transferred their attention to veterinary science, was completely ignorant of any veterinary knowledge. It is said that his sole claim to authority lay in the fact that he had dissected and described a horse's eye.

His appointment was not received well by those who had veterinary knowledge and especially by those with medical qualifications, and he met with much opposition. Safe in the post, being sponsored by J. Hunter and Cline, Coleman sought for the means to thwart opposition. He advised the governors that it was not necessary for a veterinary student to be well educated, that grooms and kennelmen would make the best veterinary surgeons and that the sons of doctors made the worst. There was no need for such a long course of study and that a few months would suffice. His advice was accepted and subsequently he passed them through his training mill in three months. The College ceased to be residential and hordes of students passed through its portals and were awarded their diplomas. It was not long before Coleman could command a strong

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following in the profession, and he was able to reign and command like an autocrat.

It is strange that the great ideals on which the College had been founded, largely due to the initiative and foresight of sagacious medical men, should have been sabotaged by one of their order. The clock of progress was put back by well over half a century. Coleman was an able man and of nimble wit, in his own interest, and one cannot deny that in the course of time he did make many additions to the sum of veterinary knowledge.

Here are extracts from the reports on two of the many experiments that Coleman made whilst investigating natural and suspended respiration. I think that these reports show that Coleman was a keen investigator and his powers of observation were acute. His book entitled "Dissertation on Natural and Suspended Respiration" was published in 1802 and dedicated to Henry Cline. It is interesting and instructive even now.

(1) "A cat was strangled, and five minutes after the last expiration the chest was opened; the lungs were then alternately expanded and collapsed for five minutes, the heart acted rather powerfully, but no alteration could be observed in the blood of its two sides; either as to quantity or quality.

"The heart was now electrified by small shocks, during the existence of the collapse, and this was continued for five minutes, when, upon examination, it was observed that its action was evidently lessened; the left side became rather more distended than before, but the blood was black in both auricles and ventricles.

"The lungs were now expanded, and the heart at this instant electrified: after two shocks had been given, they were collapsed, again expanded and electrified; and this process likewise was continued for five minutes. On examining the heart, both sides were now found less distended, their action quickened, and the blood in the pulmonary veins, left auricle and ventricle, completely florid.

"The result of this experiment not only proves the advantages of the stimulating power of electricity on the heart, beyond that of simple inflation; but also evinces the superiority of administering it in the distended, over the collapsed state of the lungs."

(2) "A cat was strangled; and after it had ceased to breathe, the body and extremities were thoroughly rubbed for ten minutes, the chest was then opened. On examining the heart, the right side was found fully distended, and the left rather more so than usual, without any sign of action in either.

"An opening was then made in the inferior cava, so as to let out a portion of blood; and the action of the right side of the heart was soon renewed.

"This experiment was repeated, and it invariably resulted that the more the right side of the heart was distended the weaker was its action, and, even where no action was evident during the distention, it was generally renewed by removing part of the blood from the anterior cava."

Coleman resisted all attempts at reformation and particularly so if those projects were sponsored by men who were products of the original three-year

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course or had medical qualifications. I am afraid that there was little integration in this country again between medical men and veterinary surgeons until 1871, by which time Coleman and his two successors, Sewell and Spooner, had departed this life.

The years between 1800 and 1871 wilted under E. Coleman's autocracy and the complacency and vacillation of his successors, Sewell and Spooner, but they were not completely sterile. In spite of the lowering of the preliminary educational standards and the debasement of the course of training, some good men were produced who kept the torch of truth and knowledge glowing.

When the Veterinary College was established, rules and regulations were ordained for the conduct of affairs. Regulation XVII stated:—

> There shall be chosen, on the day of election each year, two committees, viz.:
>
> A medical experimental committee and a
>
> Committee of Transaction.

John Hunter was appointed Chairman of the Medical Committee and amongst the members were Mr. Cline, Mr. Vaux, Sir George Baker, Dr. Packwood, Mr. Peake and Mr. Horne and Mr. Sheldon. These were all men of medical renown and Regulation XIX specified the duty of that committee.

> "The Medical Experimental Committee shall meet occasionally for the purpose of suggesting and trying experiments with a view to throwing additional light on animal economy and to discover the effects of medicines upon different animals to be procured for that purpose; and this Committee shall from time to time make reports of their proceedings to the Council."

It will be seen that the men who promulgated such a regulation were men of vision who anticipated rightly that the Veterinary College would become a great centre of learning and for the dissemination of knowledge. It might have been so from the earliest days if Coleman, probably to cover his own ignorance, had not drawn the veil of secrecy over the work of the institution. He opposed all efforts for reform, he refused to train his students on any animal other than the horse. He did not welcome veterinary surgeons into the College and refused to have them on the board of examiners. In all this stupidity he was supported by his friend, Astley Cooper. I doubt if J. Hunter would have succumbed to his persuasion. His conduct led to a justifiable reaction in the profession and this was made manifest in the columns of the "Veterinarian" under the Editorship of William Youatt, who was veterinary surgeon to the Zoological Society, and by Bracey Clark, a very competent practitioner, in "The Farrier and Naturalist," and later in the "Hippiatrist."

These periodicals helped to save the situation, for the members of the profession, outside the College, did not accept or follow the code of secrecy.

Many books and pamphlets dealing with the diseases and economy of the domesticated animals were published by veterinary surgeons and medical men. One can read to-day, and with pleasure and profit, the articles and communica-

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tions of such men as Percival, Blain, Youatt, Bracey Clark, Lawrence, Castley, Gamgee and Barlow.

During the first half of the 19th Century, largely owing to the stupidity of Coleman and his medical board of examiners, who looked upon veterinary surgeons as inferior men, relations between the two professions were not ideal. Medical men were consulted and appointed to government committees of inquiry into the causes and prevention of epidemic diseases of animals to the exclusion of veterinary surgeons. The advice of competent and experienced veterinary surgeons was ignored and with disastrous results, especially in the case of cattle plague which ravaged the country periodically.

All things come to an end, and when Simonds became Principal of the Royal Veterinary College on the death of Spooner the school entered upon a new birth. He expanded the curriculum to include the other important domesticated animals. He provided better facilities for the teaching of anatomy, physiology, pathology and chemistry, and although in his official duties as veterinary adviser to the Privy Council and Government his wise counsels on the control of epidemic diseases was often discarded in favour of those given by medical men, the seed of better relations between the two professions was sown.

If one studies the list of the teachers at the Royal Veterinary College in the late 19th and early 20th Centuries one will note the names of medical men of fame. T. S. Cobbold, D'Arcy Power, Henry Power, T. G. Brodie, George Buckmaster, Legge Symes, Marrett Tims and W. B. Bottomley. These men gave great service to the College and did much to show that medical men and veterinary surgeons belonged to one great science and on equal terms.

The same type of amicable collaboration was witnessed in other veterinary schools, medical men undertaking much of the teaching in the preclinical period. The names of Macdonald and Roaf of Liverpool, Lindsay of Glasgow and Dryerre of Edinburgh come to mind, and these men are held in high regard by the veterinary surgeons who sat at their feet.

The list of the men who have given valuable assistance to the Royal College of Veterinary Surgeons at the examinations leading to the diploma of membership would make a medical Debrett.

In the latter half of the 19th century when pathology claimed a rightful place in the curriculum and especially when bacteriology entered upon its golden days, a great new field of common interest was opened up. It is in this field that integration between the two professions was at its best for the majority of the workers were qualified men or veterinary surgeons. Two men did more than any others to bring about this fusion of interests: Sir John MacFadyean on the veterinary side and Professor William Bulloch on the medical side.

As academic knowledge of the nature of disease processes and the cause thereof accumulated, other workers were able to put it to practical application in an effort to solve and resolve problems of public health and preventive medicine. Prime workers in this field on the medical side were Delepine, Sims Woodhead,

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Leighton, Savage and in recent years Dalrymple Champneys: on the veterinary side the names of Walley, Stockman, Brittlebank, Hayhurst, Malcolm and De Vine come immediately to mind.

Up to this point, which carries one well into the present century, progress in medical and veterinary science, with a few exceptions such as Pasteur, was effected through the efforts of men who were qualified to practice those sciences, if necessary, in the field. No man can be an expert in all fields and it soon became evident that if progress was to be made one should know more and more about a little and less and less about a lot.

There was only one answer—specialisation—and this meant also that men qualified in other disciplines such as chemistry, biochemistry, physics, zoology, etc., had to be called in to solve problems which were beyond the ken of even the most able doctor or veterinary surgeon. In the medical and veterinary schools at the present day there are many men and women conducting valuable research work on diseases which they would be unable to recognise in a living patient.

These workers may have their problems as regards integration between their own specialities but from the wider view of medical or veterinary science and practice the world is as one and they are equally at home in a veterinary or medical institution.

Now this apparently salubrious outlook has brought about a state of affairs which fills me at times with some misgivings with regard to the training and subsequent competence, on graduation, of medical and veterinary students to practice the art and craft of their profession. The pure science subjects have achieved an importance in the curriculum and in the examination tests far beyond what to my mind is necessary for the production of a competent doctor or veterinary surgeon. The number of these specialist teachers in the schools is such that by their votes they can exercise a profound influence on the selection of students and upon the course of instruction which is designed to prepare the undergraduate for a career which the specialist teachers are not qualified nor competent to pursue. There is also a tendency to devote more and more time to these specialised ancillary subjects. A lot of time is spent in acquiring knowledge which a student will never be called upon to put into practice. This is to the detriment of the time and thought which can be devoted to the study of the normal and abnormal functions and reactions of the animals which they will be called upon to attend in the stable, byre and home.

In the medical sphere the cry has been heard: "Let us get back to the bedside." In the veterinary field it is just as important that we keep our feet in the stable and byre. Whilst I acknowledge fully that a knowledge of zoology, botany, chemistry, physics, biochemistry and experimental pharmacology is a necessary preliminary to the better appreciation of the vocational part of a medical or veterinary student's curriculum, I cannot accept that it is necessary for our students to delve as deeply as their colleagues who are proceeding to a degree in these disciplines. When the standard is set too high and the time

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allocated to a subject is limited, then the examination estimate of the knowledge acquired may indicate only that the student has a good or a bad memory. The examination tests become as hurdles in a race. When a hurdle is negotiated, 90 per cent of the information which has been acquired at the cost of much anxiety and toil can be discarded. This is exactly what does happen and with great rapidity.

I doubt if the majority of the students who negotiate Part I of the pre-clinical examination successfully could repeat their success after three or four months' absence from the classroom even if the written papers, the practicals and the examiners were the same. My colleagues who are responsible for these subjects tell me that it is necessary to go deeply in order to cultivate the students' powers of observation and deduction. If this be a cogent reason, then an attractive subject such as bird-watching would serve equally well. It is not necessary and we should not demand that our students become experts in any one subject. The standard should be: Is their knowledge of a particular subject sufficient to enable them to understand and appreciate subsequent studies? In the making of a good doctor or veterinary surgeon there are other things which far transcend any academic ability as measured by the results of examination tests and they are the inherent motives or qualities which caused him to choose such a career and his capacity to make use of the knowledge he has gained coupled with a reasonable amount of what is called commonsense and judgment.

These qualities will be called upon constantly every day of his professional life. He will certainly find little use for the abstruse and finer details of the various "ologies." Well over 90 per cent of the situations that he will be confronted with in practice will be ordinary or common and easily recognised and treated if he be blessed with a reasonable degree of commonsense and judgment.

In recent years whilst the schools have been in a "sellers" market as regards students it has been the custom to fill the vacant places by selection from amongst those who have attained the statutory preliminary standard of education. The tendency has been to select those who produce the educational certificates of the highest merit. I have been able to observe the undergraduate careers of about 800 students. A high preliminary educational standard, even when that is honoured by a degree, does not indicate always that they will pursue the professional course with similar success. The reverse is also the case. I have noted also that there is a tendency to favour candidates whom the selection board may think will make good research workers.

This attitude is dangerous and ridiculous, for how can one assess a candidate's aptitude to pursue a research career at a time when he has no intimate knowledge of the profession which he desires to follow?

In the first instance the medical and veterinary schools should aim to produce a graduate who is capable of doing those things which an ordinary doctor or veterinary surgeon is called upon to do in the ordinary course of practice. High academic knowledge is of no value if the young man cannot comply with this

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standard. For instance, I have personal knowledge of a highly qualified young doctor of two years' standing who failed to recognise a case of "mumps" which could have been diagnosed by a washer-woman, and of a similarly highly qualified young veterinary surgeon who thought that some Jersey cows were afflicted with jaundice because of the colour of their skin.

It is good to foster research for no profession can afford to stay in *status quo*: new sources of knowledge must be tapped constantly if we are to progress. I submit that research workers should have a reasonable knowledge and proficiency in the art and practice of their profession before they devote their whole time to such academic pursuits. If this is not the case then why should a man waste his time studying for 5 or 6 years to gain the qualification of a profession which he has no intention of practising when an ordinary degree in some scientific subject would suffice? I think that in our schools too much stress is laid on research and not enough on the teaching activities of the members of the staff. In some schools there are men and women ostensibly holding teaching posts whom the students see but rarely and in some cases have never addressed. Schools exist primarily for the instruction of students in order to fit them for a particular job, i.e., to practise the art and science of medicine or veterinary surgery. If one could abolish the practitioners, both professions would die. If research activities were to come to an end grievous harm would be done but the blow would not be mortal and the professions would still live. This undue emphasis on research has led to a curious situation. I have observed an increasing tendency to assess the worth of an individual according to the output of his publications and not by their quality or originality. I have known of occasions when the list of publications has been solemnly counted and the titles not mentioned.

Young men and women working in all branches of scientific endeavour recognise that the tally of their publications exerts a profound influence on their prospects of advancement. Their reaction is vigorous and thousands of articles which are supposed to add to the store of knowledge are published each year. The state of the world suggests that most of them serve no useful purpose.

In this address I have attempted to show you how our two professions were allied at the time of origin of the veterinary profession in this country, and that we have much in common in our daily work and our educational problems. It is said that "Wise men talk because they have something to say but fools talk because they have to say something." I hope that you do not classify me in the latter category and I beg to remind you that, in duty bound, I had to say something.

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GENERAL ARTICLES

THE USE OF IMMUNE SERUM IN THE TREATMENT OF RICIN POISONING

By E. G. C. CLARKE and J. H. JACKSON

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ACCIDENTAL poisoning in animals due to the ingestion of the seeds of castor (Ricinus sp.) has been reported frequently. It has been recorded as occurring in the ox (Anderson, 1948), pigs and poultry (Geary, 1950), sheep (Jesse, 1936), dog (McCormack, 1914), horse (McCunn, Andrew and Clough, 1945) and camel (Peck, 1942). Castor seed poisoning in man has been described (Bisham, 1903; Meldrum, 1900).

Due to the extreme toxicity of castor seeds, which contain about 1 per cent of the toxic protein ricin, mortality has frequently been high. The treatment hitherto employed in dealing with cases of ricin poisoning has been largely empirical. Anderson (1948) used chlorodyne in gruel, Geary (1950) kaolin and chalk in milk, and McCunn (1945) warmth and sedatives. Treatment of ricin poisoning in man has also been along general lines.

Ehrlich (1891) showed that an immunity to the effects of ricin is established in an animal which has been repeatedly fed with small non-lethal amounts of castor seed, and that the serum obtained from such immunised animals confers a passive immunity when injected into another animal. Since then, the production of ricin immunity has been confirmed and investigated further by many workers (Cornevin, 1897; Heymans, 1926; Hazen, 1927; Carmichael, 1926, 1929; Boquet, 1929, 1941) and the mechanism of the reaction *in vitro* between antigen and antibody has been examined extensively (Jacoby, 1901; Danysz, 1902; Moriyama, 1936, 1937; Clarke, 1954).

Forsyth (1955) mentions the use of anti-serum obtained from "hyper-immunised" animals in the treatment of cases of castor seed poisoning. The present authors, however, have been unable to trace any published record of work in this connection. The experiments described below were undertaken in order to determine whether anti-ricin serum might have any therapeutic value in cases of ricin poisoning.

Experimental Procedure

The ricin used in the experiments was prepared by the method described by Osborne, Mendel and Harris (1905). The serum was obtained from a goat which had been immunised previously with ricin from the same sample. All solutions were made up in physiological saline.

Experiment 1. One hundred mice, weight 25 to 30 mg., were divided up into groups of 10 mice each. The mice in each group were divided into 5 pairs, A, B, C, D and E. All these mice were injected intraperitoneally with ricin, the mice in Group 1 receiving 128 μg., those in Group 2 receiving 64 μg., and

so on, the dose given to each succeeding group being reduced by a half. In each case the volume injected was 0.25 ml. Prior to injection, the ricin used for each mouse in pair A was mixed with 0.05 ml. of anti-ricin serum, it having been determined previously that this amount of serum was sufficient to neutralise *in vitro* the largest dose of ricin being given in the experiment. Half an hour after the injection of ricin, the mice in each pair B received an intraperitoneal injection of 0.05 ml. of anti-ricin serum. The mice in each of the C pairs

TABLE I

	AMOUNT OF RICIN INJECTED PER ANIMAL	PAIR A		PAIR B		PAIR C		PAIR D		PAIR E	
GROUP 1	128 µg	S	S	S	S	D 12	D 12	Dx 12	Dx 12	D 12	D 6
GROUP 2	64 µg	S	S	S	S	D 12	D 12	Dx 12	Dx 12	D 6	D 6
GROUP 3	32 µg	S	S	S	S	D 12	D 12	Dx 12	Dx 12	D 12	D 12
GROUP 4	16 µg	S	S	S	S	D 12	D 12	Dx 12	Dx 12	D 12	D 12
GROUP 5	8 µg	S	S	S	S	D 36	S	D 24	D 48	D 12	D 12
GROUP 6	4 µg	S	S	S	S	S	S	D 24	D 48	D 48	D 48
GROUP 7	2 µg	S	S	S	S	S	S	D 48	D 48	D 12	D 24
GROUP 8	1 µg	S	S	S	S	S	S	D 72	D 120	D 48	D 96
GROUP 9	0·5 µg	S	S	S	S	S	S	S	S	S	S
GROUP 10	0·25 µg	S	S	S	S	S	S	S	S	S	S
TIME INTERVAL BETWEEN INJECTION OF RICIN AND INJECTION OF SERUM		NIL		½ HR.		6 HRS		24 HRS		NO SERUM GIVEN	

S = a survival. D = a death.
The figures indicate approximately the number of hours between injection of ricin and death.
X = died before injection of serum.

received a similar injection after 6 hours, and those in the D pairs after 24 hours. In each case the serum was diluted with physiological saline to a total volume of 0.25 ml. The E pairs received no serum and served as controls. The results of this experiment are set out in Table I.

Experiment 2. The work of Isaacs and Grant (1927) showed that an immunity to ricin is very rapidly established if the ricin is injected subcutaneously. It was thought, therefore, that immune serum might prove more efficacious if administered by this route. An experiment similar to the last was carried out, with the difference that, while the ricin was introduced intraperitoneally, the

serum was now injected subcutaneously. Seven groups of eight mice each were used, the mice in each group being divided into four pairs, F, G, H and I. As before, the F pairs received 0.05 ml. of immune serum half an hour after the administration of the ricin, the G pairs after 6 hours, and the H pairs after 24 hours. The I pairs received no serum. The results of this experiment are shown in Table II.

TABLE 2

	AMOUNT OF RICIN INJECTED PER ANIMAL	PAIR F		PAIR G		PAIR H		PAIR I	
GROUP 1	64 µg	D 48	D 48	D 12	D 12	Dx 12	Dx 12	D 6	D 6
GROUP 2	32 µg	D 72	D 72	D 12	D 12	Dx 12	Dx 12	D 12	D 12
GROUP 3	16 µg	S	S	D 12	D 12	Dx 12	Dx 12	D 12	D 12
GROUP 4	8 µg	S	S	D 36	D 48	D 36	D 36	D 12	D 12
GROUP 5	4 µg	S	S	D 48	D 96	D 36	D 36	D 12	D 12
GROUP 6	2 µg	S	S	S	S	D 48	D 48	D 48	D 96
GROUP 7	1 µg	S	S	S	S	D 72	S	D 72	D 96
TIME INTERVAL BETWEEN INJECTION OF RICIN AND INJECTION OF SERUM		½ HR		6 HRS		24 HRS		NO SERUM GIVEN	

S = a survival. D = a death.
The figures indicate approximately the number of hours between injection of ricin and death.
X = died before injection of serum.

Experiment 3. In order to simulate more closely the conditions which exist in cases of accidental poisoning by castor seeds, it was necessary to investigate the value of immune serum in mice in which ricin had been introduced into the alimentary tract. Forty mice were used, each receiving 0.25 ml. of a 10 per cent solution of ricin in distilled water, i.e., 25 mg. of ricin. This was introduced directly into the stomach by placing the mice under light ether anæsthesia and passing a thin polythene tube, connected to the needle of a 1 ml. hypodermic syringe previously filled with ricin solution, down the œsophagus. These mice were divided into four groups, J, K, L and M, each group consisting of ten mice. Half an hour after the administration of the ricin, each mouse in group J received an intraperitoneal injection of 0.25 ml. of immune serum, this being the largest quantity which it was found to be practicable to give. The mice in groups K

and L received an exactly similar injection of serum given by the same route after intervals of 6 hours and 24 hours respectively. The mice in group M received no serum and served as controls. The results of this experiment are shown in Table III.

TABLE 3

GROUP	NUMBER OF MICE IN EACH GROUP	DOSE OF RICIN ADMINISTERED PER OS	VOLUME OF SERUM INJECTED INTRA-PERITONEALLY	TIME INTERVAL BETWEEN ADMINISTRATION OF RICIN AND INJECTION OF SERUM	NUMBER OF DEATHS OCCURRING ON :-				PERCENTAGE SURVIVAL
					1ST. DAY	2ND. DAY	3RD DAY	4TH DAY	
J	10	25 mg.	0·25 ml.	½ HOUR	1	0	0	0	90%
K	10	25 mg.	0·25 ml.	6 HOURS	0	4	0	1	50%
L	10	25 mg.	0·25 ml.	24 HOURS	2	4	4	0	0%
M	10	25 mg.	NIL		4	2	2	1	10%

Discussion

Table I shows that, where both ricin and serum were injected intraperitoneally, 0.05 ml. of serum provided complete protection against 128 times a lethal dose of the toxin, either when the serum was mixed with the ricin immediately before injection, or when administered up to half an hour after the ricin. However, when the serum was injected after an interval of 6 hours, protection was afforded against the effects of only four to eight times the lethal dose, whereas, if the injection of serum was delayed for 24 hours no protection at all was provided.

In those cases where the ricin was injected intraperitoneally and the serum subcutaneously (Table II) the serum was less efficacious than when injected directly into the peritoneal cavity. In this case, injection of 0.05 ml. of serum after an interval of half an hour neutralised only 16 times a lethal dose of ricin, and only twice a lethal dose if the serum was withheld for 6 hours. Serum given by this route 24 hours after the ricin had no protective effect. It may be that the decrease in the protective value of the serum in these cases is due to the fact that, when both serum and ricin are administered by intraperitoneal injection, some neutralisation of the toxin takes place in the body cavity before it is absorbed. On the other hand, the discrepancy may be due to varying rates of absorption of ricin and serum from different sites. The whole question of the rate and method of absorption of ricin is being investigated further, but this presents some difficulty in that it has not been possible to determine the presence of ricin in the tissues or body fluids of an animal, the method used by Clarke (1953) for the identification of ricin in plant material not being applicable.

It will be seen that the intraperitoneal lethal dose of ricin for the mouse is between 1.0 μg. and 0.5 μg. In the third experiment, however, where ricin was introduced directly into the stomach, a vastly increased dose was used. Ehrlich (1891) showed that ricin was much less toxic to guinea-pigs and rabbits when introduced *per os* than when injected subcutaneously. Our experiments

showed that in the mouse a dose of no less than 25 mg. (i.e., 25,000 times the intraperitoneal lethal dose) had to be given by mouth in order to obtain a mortality of approximately 100 per cent.

In those cases where this large dose of ricin was introduced directly into the stomach and was followed by an intraperitoneal injection of serum, our results (Table III) showed that, where the serum was given half an hour after the ricin, nine out of the ten mice treated in this manner survived. When, however, administration of serum was delayed for 6 hours, only 50 per cent of the mice survived, and when delayed for 24 hours it had no protective effect.

We have not attempted to carry out a complete histological investigation of the tissue changes induced by ricin, but our preliminary studies indicate that a lethal dose of ricin in the alimentary canal is followed by progressive destructive changes in the gut wall, most marked in the duodenum, but extending from the pylorus along the full length of the small intestine, and involving also the cæcum. Macroscopically the gut shows either punctate or diffuse hæmorrhagic areas particularly in the duodenum: histological examination shows a gradual destruction of gut epithelium, particularly over the tips of the villi, followed by a degradation of the sub-epithelial connective tissue and enlargement and subsequent rupture of the capillary blood vessels in this region. Berkeley (1896) on the other hand has described degenerative changes in neurons in the cerebral cortex due to the action of ricin: he also pointed out that the time taken for the lethal effects of ricin to manifest themselves is not dependent solely on the amount of ricin but is also dependent on the length of time that the toxin is in contact with the body tissues. The results of our experiments indicate that in the case of mice, using a massive dose of ricin, 24 hours after its introduction into the stomach irreparable tissue damage has been induced, and that for the serum to be effective it must be given as early as possible.

We have not attempted to determine the relative protective value of serum where the ricin and serum are introduced by other alternative routes. Nevertheless, the experiments described suggest that immune serum might have some value in the treatment of cases of castor-seed poisoning, provided that it can be administered quickly enough.

Acknowledgments

We wish to express our thanks to Professor E. C. Amoroso and Professor J. McCunn for the interest they have taken in this work. We are also much indebted to Dr. F. Bell for immunising the goat that provided the serum used in the experiments, and to Dr. D. G. Harvey for help in the administration of ricin *per os*. We are also grateful to Miss A. Stanley, Miss M. Boulding and Mr. J. B. Edwards for technical assistance.

Summary

Experiments are described which show that the effects of ricin poisoning in mice may be counteracted by the prompt administration of immune goat serum.

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SOME CLINICAL AND SEROLOGICAL OBSERVATIONS ON AN OUTBREAK OF CONTAGIOUS PLEUROPNEUMONIA IN A DAIRY HERD OF ZEBU CATTLE IN NIGERIA

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Bovine pleuropneumonia is an acute, subacute, or chronic contagious disease of cattle characterised by extensive œdema of the inter-lobular and alveolar tissues of the lungs, and by serofibrinous pleurisy. The disease has been recognised by veterinary officers in Nigeria for the past 35 years, and Birkett (1951) states that " its gregarious place in the disease hierarchy has become more and more prominent in recent years."

There has been little opportunity in the past in Nigeria, prior to the rapid field test recently developed by Newing and Field (1953), to combine continued clinical observations on outbreaks of pleuropneumonia in the field with laboratory diagnostic aids and post-mortem findings. During the small outbreak at University College, Ibadan, described in this paper, it has been possible to keep the herd under constant observation and to send serum samples for the complement-fixation test to the Veterinary Research Laboratory, Vom, Nigeria. The causative organism was isolated by Harper (1950), at Ibadan, although this strain was unfortunately lost.

The foundation stock for the dairy herd at Ibadan had been sent by rail from the Government Stock Farm at Shika, Northern Nigeria, in May, 1950. They arrived in good condition and were not known to have had any direct contact with other animals during the journey. All were reported to have been vaccinated against rinderpest and pleuropneumonia while at Shika, although there was no information available as to the exact date of the pleuropneumonia vaccination. The herd was subsequently maintained in strict isolation at Ibadan.

During June, 1950, 12 animals died or were slaughtered after spraying with an arsenical dip. The history of this accident is obscure but it is believed that nearly half the herd had been severely " scalded " by over-exposure to the spray, followed by excessive sunlight. The rest of the herd, which included 2 bulls and 16 cows, heifers and calves, appeared quite unaffected and continued in good health and production until September 4, 1950, when the first case of pleuropneumonia occurred.

Of a total herd of 18 animals, 9 died of pleuropneumonia, or were slaughtered, 3 recovered and the remainder appeared unaffected. The case records and laboratory reports of the animals involved in the outbreak are given at the end of the paper.

The outbreak lasted from September 4 until November 23, 1950. No further cases have occurred for a period of 5 years following the outbreak and

the herd has remained in good health and production. Of the 18 animals involved, 9 (50 per cent) died or were slaughtered, 3 (16.6 per cent) showed only mild or non-specific symptoms, and 6 (33.3 per cent) were not apparently affected.

Both peracute and acute forms of the disease were observed, death occurring in from 4 to 15 days. The typical chronic form described by Henning (1949) and by Turner (1954), in which apparently recovered animals may again break down at a later period, did not occur. What would appear to be an unusual course is that of case 5, in which clinical symptoms were continuous for 47 days before the animal died.

In most cases the onset of symptoms was accompanied or preceded by a rise in temperature, although in cases 2 and 9 there were anorexia and a slight cough almost a week before fever occurred.

The course of the disease in severe cases in pregnant animals appeared to have no effect on the normal development of the fœtus (cases 2 and 5), one live and apparently healthy calf, in fact, being removed from dam 5 on autopsy. The calf was about two weeks premature and died two days later through apparent inability to digest its food. No lesions were found. It is interesting to note, however, that Purchase (1939), cited by Henning (1949), has reported eye and joint lesions in a calf born of a dam which had just recovered from pleuropneumonia.

Complement-fixation tests were made during the outbreak on blood samples drawn from the jugular vein of 13 animals on October 5, 1950. All survivors (except calves, cases 17 and 18) were then vaccinated* against pleuropneumonia on October 8, 1950, and subsequently on February 23 and May 15, 1951, and January 2, 1952, but these vaccinations are thought to have had little or no effect.

All animals were negative to the complement-fixation test on March 19, 1952, but there was some reaction to the slide agglutination test in cases 3, 6, 7, 13, 14 and 15, probably due to vaccination in January, 1952. All were negative to both tests on November 15, 1952, except for a trace reaction to the complement-fixation test in cases 6 and 10.

One calf, born of a dam with a positive titre to the complement-fixation test (case 6), gave a negative test soon after birth.

An analysis of the table (Fig. 8) shows that of the 7 animals (cases 4, 7, 8, 9, 13, 14 and 15) *negative* to the complement-fixation test on October 5, 1950, 5 (70 per cent) showed no symptoms of pleuropneumonia and 2 (30 per cent) died. Of the 2 animals which died, however, 1 (case 9) was tested only one day after the onset of symptoms, while the other (case 4), except for a transient fever, showed no definite symptoms until a week after the test.

Of the 6 animals (cases 2, 3, 5, 6, 10 and 11) *positive* or *doubtful* to the complement-fixation test on October 5, 1950, 5 were tested after the onset of symptoms and of these 2 (33.3 per cent) were later slaughtered on the point of death, 3 (50 per cent) recovered after showing mild or non-specific symptoms, and the other 1 (16.7 per cent) showed no symptoms.

*Khartoum strain, Vom vaccine, living Bennett's broth culture, 85th generation (approx.).

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Although the complement-fixation tests were made on a very limited number of animals, these results agree in general with those of Hindmarsh, *et al.* (1943) who observed that serum is unlikely to be positive to the test when taken during the incubation period, but positive if drawn from animals showing symptoms of pleuropneumonia. They also state that cattle recovering from the disease will give a positive, but gradually declining, titre as recovery progresses. In cases 3 and 10, both animals showed only transient or non-specific symptoms and retained a positive titre for 12 months.

The origin of the outbreak is uncertain, but it is suggested that there could have been a "lunger" in the herd which, following the accident with the arsenical dip in June, 1950, might have broken down with pleuropneumonia, thus disseminating infection among the rest of the herd. The fact that the first case of pleuropneumonia was not observed until September, 1950, might be explained by the long incubation period which may elapse before symptoms appear (Turner, 1954).

Control measures included the slaughter of clinically affected animals, isolation of positive reactors, and vaccination and segregation of apparently non-infected animals. Positive reactors which did not later break down with the disease were also vaccinated and isolated for two years in a pen 300 yards from the main byres, before rejoining the herd.

All animals, including calves over 2 weeks old, have been vaccinated biannually since the outbreak as a routine measure, although, as Mahoney (1954) in Australia has stated, calves may receive little protection from pleuropneumonia when vaccinated below the age of 8 months. The systemic reactions which have been reported by Turner (Mahoney, 1954; A.V.A. Conference—Discussion) in young calves following vaccination were not observed at Ibadan, no doubt due to the high degree of attenuation of the vaccine used (Gambles, 1955).

Summary

An outbreak of contagious pleuropneumonia in a dairy herd of zebu cattle is described. The results of the complement-fixation test are discussed in relation to clinical and post-mortem findings. Methods used to control the outbreak are given.

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CASE HISTORIES

Case 1. Bull. No complement-fixation test performed.*

Symptoms. Dyspnœa, anorexia and fever (104.8° F.) on morning of September 4, 1950. Breathing became progressively laboured, terminating in a short grunt, although there was no cough. Four days later the temperature was 98, the pulse rapid and weak, and it died at noon.

Lesions. The thoracic cavity contained several quarts of fibrinous exudate. The left lobes of the lungs appeared normal, but the right cardiac and apical lobes showed extensive areas of fibrinous pleurisy and adhesions, marbling and red hepatisation. A clear, yellow liquid could be squeezed from the interstitial tissue of the lung parenchyma and bronchioles. The bronchial lymph glands were swollen and wet on cutting. There was slight pericarditis and the heart was thin and wedge-shaped, having become sandwiched between firm, cheese-like masses of exudate in the mediastinum. Except for slight inflammation of the abomasal mucosa all other organs and tissues appeared normal.

Case 2. Heifer. October 5, 1950. The complement-fixation test was strongly positive at 1 : 2,500 (approx.).

Symptoms. Anorexia and listlessness two weeks before rise in temperature and dyspnœa on September 23, 1950 (Fig. 1). By the evening of October 6, paroxysms of coughing and grunting, accompanied by pleuritic raspings, audible 3 feet away, were the predominant symptoms. There was slight œdema of the eyelids. Although anorexia was complete the animal drank almost up to the time of death. Slaughtered on the point of death at 9.30 a.m., October 8.

Lesions. Massive fibrinous pleurisy and adhesions of both lungs, and extensive consolidation and marbling of all lobes, with little normal parenchyma remaining. The thoracic cavity and lung parenchyma, from the sternum to the dorsum of the mediastinum, were invaded by a solid mass of fibrinous exudate. There was slight inflammation of the mucosæ of the abomasum and colon. All other organs and tissues appeared normal. The uterus contained a 4½ months fœtus.

Case 3. Bull.

C.F. test, October 5, 1950. Serum anticomplementary.
" " November 11, 1950. Doubtful. Partial fixation at 1 :10.
" " September 14, 1951. Negative.
" " March 19, 1952. Negative. (Slight reaction to slide agglutination test.)
" " November 15, 1952. Trace. (Negative to slight agglutination test.)

Symptoms. This animal did not show typical symptoms of pleuropneumonia. There were intermittent attacks of diarrhœa, fever and anorexia from September 4, 1950, to November 15, 1950, but no distressed breathing or coughing. Piroplasmosis was diagnosed on October 30, 1950, and complete recovery followed treatment with Babesan.

Case 4. Cow. C.F. test, October 5, 1950. Negative.

Symptoms. The temperature rose to 103° F. on the evening of September 27, 1950. Distressed breathing, anorexia, cessation of rumination, bloat and coughing were observed on October 13, 1950. The animal died from apparent asphyxia on the evening of October 28, 1950.

Lesions. Extensive fibrinous pleurisy and adhesions, massive areas of consolidation of lung parenchyma and effusions of serous exudate into the thoracic cavity. Slight œdema in the umbilical fold.

* The complement-fixation test used was according to the technique described by Campbell and Turner (1936) and Campbell (1938), with minor modifications adapted from Hole and Coombs (1947). The use of this test in Nigeria has previously been referred to by Newing and Field (1953).

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Case 5. C.F. test, October 5, 1950. Doubtful. Almost complete at 1 :10. Partial fixation at 1 :40.

Symptoms. The disease took a chronic or protracted course. There was a rise in temperature on the evening of September 15, 1950 (Fig. 4), followed on October 16, 1950, by distressed breathing, coughing and diarrhœa. These symptoms abated slightly until early October, but became progressively severe after this, until the animal was slaughtered on the point of death on November 2, 1950.

Lesions. Extensive fibrinous pleuritis and adhesions, with almost complete consolidation of all lobes of the lungs. Immediately after slaughter a live and apparently healthy calf was removed by laparotomy. The dam was about two weeks from completion of normal term of pregnancy. This calf died two days later from what appeared to be a digestive disorder.

Case 6. Cow.

C.F. test, October 5, 1950. Strongly positive. Complete fixation at 1 :40. Partial fixation at 1 :80.
" " July 7, 1951. Partial fixation at 1 :10.
" " September 27, 1951. Negative.
" " March 19, 1952. Slight reaction to slide agglutination test.
" " November 15, 1952. Trace only. Negative to slide agglutination test.

Symptoms. Other than for fluctuations in temperature from October 13, 1950, to October 26, 1950 (Fig. 2), and slight diarrhœa on October 13, 1950, and November 11, 1950, this animal showed no symptoms of pleuropneumonia and has remained in good health and production.

Case 9. Cow. C.F. test, October 5, 1950. Negative.

Symptoms. A cough was first noticed on October 4, 1950, but there was no fever until October 10, 1950 (Fig. 3). Distressed breathing, coughing, anorexia and intermittent bloat became progressively worse until the animal died in a state of extreme bloat on the evening of October 19, 1950.

Lesions. Resembled those described in Case 1.

Case 10. Heifer.

C.F. test, October 5, 1950. Strongly positive at 1 :1,280.
" " July 7, 1951. Complete fixation at 1 :20.
" " September 27, 1951. Doubtful. Partial fixation at 1 :10.
" " March 19, 1952. Negative. Negative to slide agglutination test.
" " November 15, 1952. Trace (probably post-vaccination reaction). Negative to slide agglutination test.

Symptoms. This heifer showed no apparent symptoms of pleuropneumonia. Fluctuations in temperature between November 1, 1950, and November 26, 1950, were ascribed to infection from *Trypanosoma vivax,* diagnosed from stained blood smears on November 15, 1950.

Case 11. Cow. C.F. test, October 5, 1950. Doubtful. Serum anti-complementary. Some fixation at 1 :20. Partial fixation at 1 :40.

Symptoms. This was the only case in which apparent recovery occurred after symptoms of coughing had once become established. There was an intermittent cough and rise in temperature (Fig. 5) on September 5, 1950. The temperature returned to normal after October 21, 1950, but a slight cough continued till early November, 1950. She aborted in February, 1951, but the cause of this was not ascertained. Slaughtered at the end of February, 1951, for investigational purposes, but no lesions were found.

Case 12. Bull. No C.F. test performed.

Symptoms. An acute case characterised by a sharp rise in temperature on October 16, 1950 (Fig. 6), and progressively severe symptoms of anorexia, dyspnœa, slobbering and coughing. Death occurred from apparent asphyxia on October 28, 1950.

Lesions. Resembled those described in Case 1.

Case 16. Cow. No C.F. test performed.

Symptoms. A peracute case characterised by high fever on September 5, 1950 (Fig. 7), distressed breathing, coughing and anorexia. Died September 14, 1950. No post-mortem examination.

Cases 17 and 18. Calves, 5 months of age. No C.F. test performed.

Symptoms. Both calves showed symptoms of fever, dyspnœa and coughing on or about November 15, 1950, and died on November 22, 1950, three weeks after the end of the outbreak in the adult herd.

Lesions. Extensive fibrinous pleurisy and adhesions, consolidation and red hepatisation of about 50 per cent of the lung parenchyma and copious exudates in the thoracic cavities.

Cases 7, 8, 13, 14 and 15. Cows. C.F. tests all negative. No symptoms of pleuropneumonia.

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FIG 1

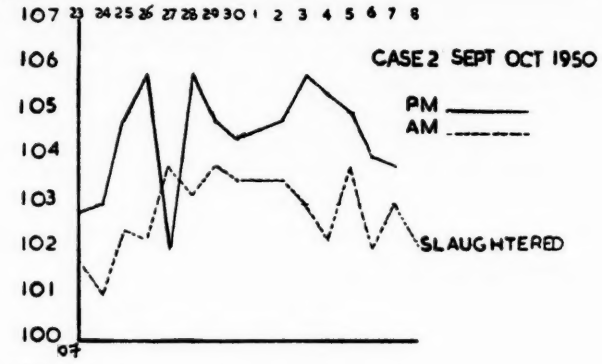


FIG 2

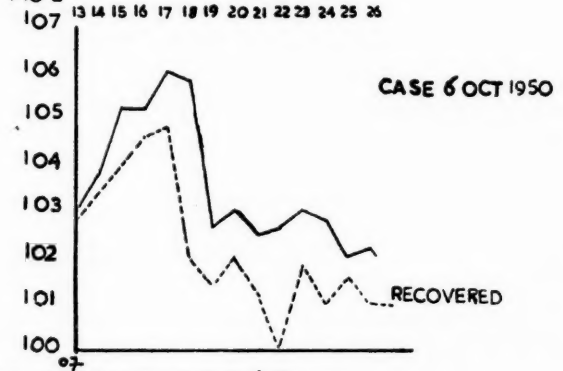
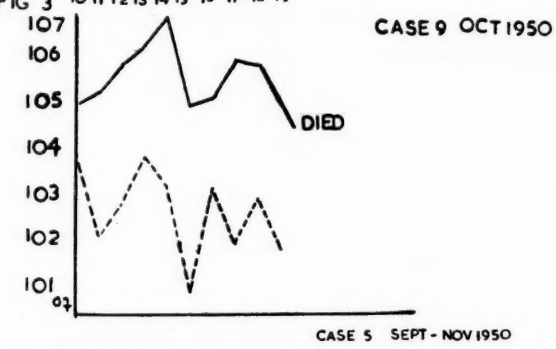


FIG 3



CASE 5 SEPT - NOV 1950

FIG 4

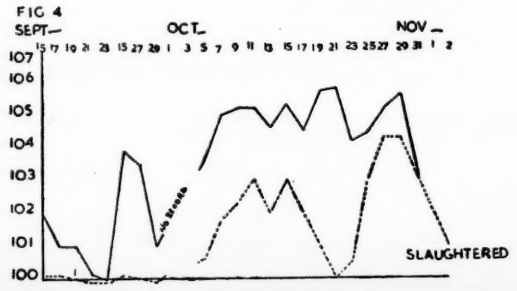


FIG 5

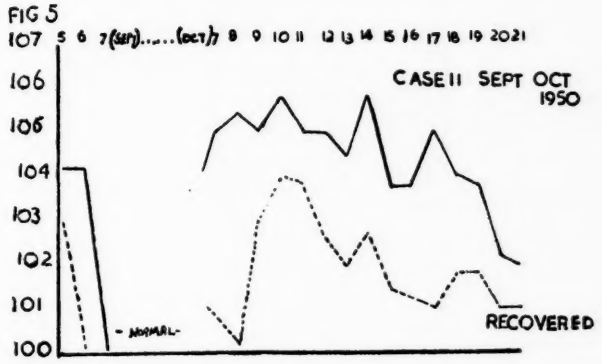


FIG 6

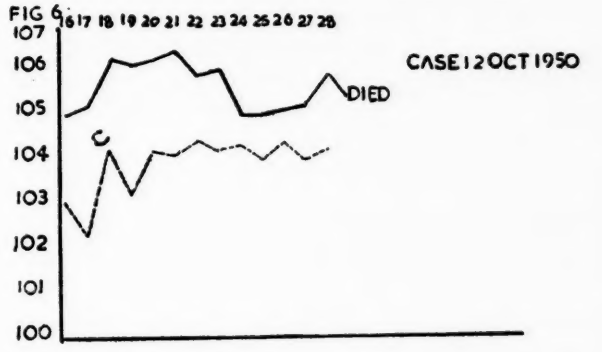


FIG 7

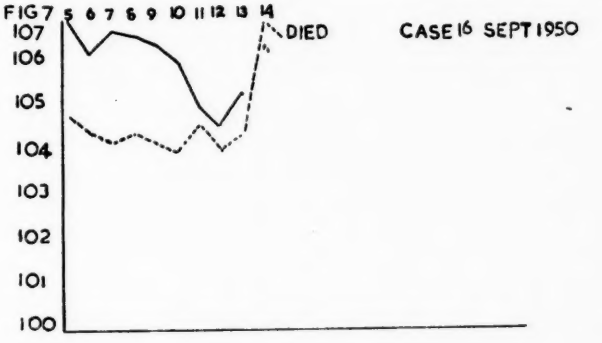


FIG. 6. RESULTS OF SEROLOGICAL TESTS AND CLINICAL FINDINGS.

Case No.	Sex	Test	Date and result		Date of rise in temperature	Date of onset of clinical symptoms	SEQUELAE	REMARKS
1	M	Not done	-					
2	F	CF test	5.10.50.	Strongly positive 1:2,560 approx.	4. 9.50	4. 9.50	Died 8. 9.50	Peracute form.
3	M	" "	5.10.50.	Serum anti-complementary	23. 9.50	16. 9.50	Slaughtered 7.10.50	Acute form. Slaughtered on point of death.
		" "	7.11.50.	Doubtful. Partial fixation at 1:10				
		" "	19. 3.52.	Negative	4. 9.50	4. 9.50	Recovered	Symptoms transient and non-specific
		Slide ag.	"	Some reaction				
		" "	15.11.52.	Negative				
		CF test	"	Trace				
4	F	CF test	5.10.50.	Negative	27. 9.50	13.10.50	Died 28.10.50	Acute form.
5	F	CF test	5.10.50.	Doubtful. Incomplete at 1:10				
				Partial " 1:40			Slaughtered 2.11.50	Protracted form. Slaughtered on point of death
					15. 9.50	16. 9.50		
6	F	CF test	5.10.50.	Strongly positive. Complete fix. 1:40				
				Partial " 1:80				
		" "	7. 7.51.	Partial " 1:10	13.10.50	None or slight	Recovered	Symptoms indefinite. Calved 4. 4.51. Calf negative to CF test.
		" "	27. 9.51.	Negative				
		" "	19. 3.52.	Negative				
		Slide ag.	"	Some reaction				
		" "	15.11.52	Negative				
		CF test	"	Trace				
7,8	F's	CF test	5.10.50	Negative	-	None		No symptoms whatever.
		" "	7. 7.51	Negative				
		" "	19. 3.52	Negative				
		Slide ag.	"	Some reaction (Case 7 only)				
		" "	15.11.52	Negative				
		CF test	"	Negative				
9	F	CF test	5.10.50	Negative				
10	F	CF test	5.10.50	Strongly positive at 1:1,280	10.10.50	4.10.50	Died 19.10.50	Acute form
		" "	7. 7.51	Complete fixation " 1:20	1.11.50	None	-	Mild trypanosomiasis 15.11.50
		" "	27. 9.51	Partial " 1:10				
		" "	19. 3.52	Negative				
		Slide ag.	"	Negative				
		" "	15.11.52	Negative				
		CF test	"	Trace				
11	F	CF test	5.10.50	Doubtful. Serum anti-complementary				
				Some fixation at 1:20				
				Partial " 1:40	5. 9.50	5. 9.50	Recovered	Slaughtered for investigational purposes. No lesions.
12	F	Not done	-					
13,14,15	F's	CF test	5.10.50	All Negative	16.10.50	16.10.50	Died 26.10.50	Acute form
		" "	27. 9.51	" Negative	-	None		No symptoms whatever
		" "	19. 3.52	" Negative				
		Slide ag.	"	" Showed some reaction				
		" "	15.11.52	" Negative				
		CF test	"	" Negative				
16	F	Not done	-		5. 9.50	5. 9.50	Died 14. 9.50	Per. Acute form
17,18	Calves	Not done	-		No record	15.11.50 (about)	Died 22.11.50	Acute form

Note — All survivors vaccinated 8.10.50, 23.2.51, 15.6.51 and 2. 1.52. (except calves 17 and 18)

(Tabulation of preceeding data.)

THE PREPARATION OF EXPERIMENTAL CONCENTRATED FREEZE-DRIED CONTAGIOUS BOVINE PLEUROPNEUMONIA VACCINE

By R. W. WHITE

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PRIESTLEY (1955*a*, *b*) has described the results of immunising cattle with freeze-dried *Asterococcus mycoides* organisms incorporated in agar. These results were so promising that mass production of the vaccine was started in Khartoum in November and December, 1954, with a view to carrying out an extended field trial. Since this type of vaccine is likely to come into general use in the Sudan, it has been considered worth while to describe the technique of its preparation in some detail.

The vaccine consists of young cultures of *A. mycoides*, concentrated centrifugally, resuspended in 50 per cent serum, and vacuum freeze-dried in 0.1 ml. volumes in ampoules. The reconstituted material is mixed with 1 per cent agar solution immediately before injection into cattle.

Preparation

Cultures.

The basic culture medium used was Bennett's (1932) ox heart infusion broth with 2 per cent Difco peptone in one-litre flasks containing approximately 900 ml. each.

Horses were bled aseptically and serum drawn off from the clot, with sterile precautions, bottled in 50 ml. quantities and refrigerated. A bleeding yielded approximately 750 ml. of serum. The first and last bottle from each bleeding were tested for sterility at the time of bottling, by sowing 1 ml. into 9 ml. broth and incubating this for a week. If either bottle showed contamination, all the bottles from that particular batch were then tested.

For seed, a bank of 10th generation cultures of *A. mycoides* had been prepared and stored in a deep-freeze cabinet (Frigidaire Conservator JC26). From a tube of this, a few tubes of 48-hour-old cultures in serum broth were prepared for sowing each batch of flasks.

Seeding.

To 25 bottles of 50 ml. serum was added crystalline "G" penicillin, freshly dissolved in broth, at the rate of 4,000 units per ml. (White, 1954, 1955), and each bottle of serum poured into a flask of broth. About 1.5 ml. of seed was added, the flasks shaken and incubated for two days. Out of 200 flasks of culture thus sown, only 5 became contaminated and 1 failed to grow.

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The culture seed was not added to the undiluted serum because of the possibility that this may affect virulence of the culture.

The unit of 25 flasks of culture constituted one day's run in all subsequent operations, and therefore yielded one batch of vaccine. This amount was the maximum that could be handled satisfactorily in any one day.

Centrifugation.

After the 2 days' incubation, films stained by stevenel blue (Priestley, 1952) from all flasks were examined, and the cultures concentrated in a Sharples tvpe No. 1A electric drive centrifuge.

The maximum motor speed of this centrifuge is 12,000 r.p.m. (the ratio of motor to bowl speed is approximately 1 to 2). In order to overcome voltage drop experienced in the local mains supply, an auto-transformer (Claude Lyons and Co.), with input tapped at 210 volts and output at 240 volts, was inserted. Control of speed was retained by means of the normal centrifuge motor rheostat, and a hand tachometer (Hasler Telegraph Co.) was applied to the motor spindle at frequent intervals. Motor speeds actually recorded varied between 11,500 and 12,200 r.p.m. The Sharples clarifying bowl (or rotor), open type, was used, the organisms remaining on the inside surface at the end of a run. The outflow is mainly from the upper bowl cover when using this type of bowl.

The culture was strained through muslin and fed to the centrifuge through rubber tubing with a screw clip from a funnel 8 inches in diameter, mounted 3 feet above the centrifuge inlet. Before a run, the centrifuge bowl, drag assembly (bowl bottom bearing), feed nozzle and bowl covers, together with the feed funnel, rubber tubing and muslin for straining the culture, were all steamed for 30 minutes.

The culture was put through the centrifuge in lots of 12 or 13 flasks (10,800 to 11,700 ml.) each, two such lots being handled each day. The average time for a run was 106 minutes, the measured throughput volume average (from the upper bowl cover only) was 82 ml. per minute.

Harvesting and Resuspending the Organisms.

At the end of a centrifuge run, and after removal of the bowl bottom, the concentrated organisms were immediately harvested from the bowl in 4 to 6 ml. sterile distilled water by repeated washing, using a teat controlled pasteur pipette. 0.5 ml. of this suspension was rapidly titrated by serial dilution in saline, against opacity tubes, and the bulk brought to No. 30 (equivalent) on the Brown's Opacity Scale with distilled water. At once, an equal volume of sterile inactivated (56° C. for 30 minutes) horse serum was added. The final volume of suspension obtained in any one day varied between 41.2 and 132.6 ml. There was a tendency for the volume of finally standardised suspension obtained from each batch to increase as the work proceeded, which could not be accounted for. It may be noted that the washing of the organisms on the spinning bowl by feeding through saline (or broth) at the end of a centrifuge run, as described by Campbell and

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Turner (1953) in the preparation of antigen, was deliberately not done. Variations in the serum, therefore, might have given rise to a variable opacity reading. Detailed records of speeds, throughput volumes, and any adjustments necessary during centrifugation were kept, and examination of these failed to reveal any factor responsible for the variation noted.

A total of eight batches of material were handled in this and all subsequent operations.

Distribution of the Suspension to Ampoules.

This was done the same day that the suspension was prepared. An automatic pipetting unit (A. L. Hawkins & Co.), as is employed for repetitive delivery in serological tests, was used. Such a filler is not claimed to deliver the final 0.1 ml. accurately, and it was found necessary to re-calibrate this.

In order to reduce the priming volume, the rubber tube carrying the pick-up nozzle was shortened to 11½ inches, this being the least length still allowing sufficient play for the filler with the cannula fitted to operate vertically over ampoules. The priming volume was then 3.5 ml.

Immediately before use, the ampoule filler was sterilised in boiling water. The suspension, the pooled product of two centrifuge runs, was filled out in 0.1 ml. amounts in sterile ampoules 7.5 x 90 mm., the cotton-wool plugs being discarded, and the racks of filled ampoules covered with a layer of sterilised muslin during all subsequent handling (except when under vacuum). Filling was carried out under a large hood. After filling, the ampoules were stored in the deep-freeze cabinet at —20° C. overnight.

Circular racks of light gauge metal, and of a size to fit the dessicators in which secondary drying was done, and holding 202 ampoules, were made locally.

Freeze Drying.

The ampoules filled out from the previous day's run were removed from the deep-freeze cabinet, allowed to melt, and freeze-drying carried out.

The freeze-drying apparatus consisted of a bench model (Edwards & Co., type L5) centrifugal machine, which has the Speedivac type 2S2 pump. A drying chamber in which an additional header port and connection had been fitted was obtained, also a centrifuge carrier plate specially drilled to take 48 ampoules 7.5 mm. diameter, instead of the usual mixed load carrier plate.

The ampoules with suspension were racked up, 48 at a time, in the special carrier plate, and each complete load was spin frozen over phosphorus pentoxide in the machine, and allowed to dry for 45 minutes. A pressure of 0.3 to 0.2 mm. mercury was obtained in about 2 minutes and maintained throughout.

The modification of the McLeod gauge known as the Vacustat (Edwards and Co.) was used for pressure measurement. One port of the drying chamber of the machine was fitted with a T-piece having a secondary header screw connection on one arm) which was blanked off during primary drying), and a nozzle for rubber tubing on the other arm. From this nozzle arm a connection was made

with pressure tubing to a metal Y-piece, and from this, one connection made to the Vacustat permanently. The other arm of the Y-piece was used for connection to a dessicator as described below, and was blanked off with glass rod when not required. The second port of the drying chamber was also blanked off during primary drying.

At the end of the 45-minute drying, the ampoules of partially dried vaccine were removed from the machine, and transferred from the carrier plate to a large rack in a dessicator, over phosphorus pentoxide; and the dessicator evacuated immediately. The trays of dessicant in the machine were changed, a fresh run of 48 ampoules was immediately set going, and the whole process repeated until all ampoules had been frozen and transferred to dessicators under a vacuum of 0.3 to 0.2 mm. of mercury. A spare set of dessicant trays permitted rapid reloading between runs. The dessicators were stored in the refrigerator for secondary drying to proceed, for at least 10 days. Pyrex-glass vacuum dessicators were used.

Sealing the Ampoules.

As indicated earlier, the drying chamber of the freeze-drying machine was fitted with an additional header port. When sealing the ampoules, three standard drying headers, each with rubber nozzles to take 30 ampoules, were mounted on the drying chambers in the following manner. The T-piece having a nozzle arm joined to the Vacustat gauge as described, was left in position, and a second T-piece, which was fitted to take a drying header screwed on both arms, was added to it in place of the blanking-off cover. On this second T-piece, two drying headers were mounted. The third drying header was attached to the spare chamber port, and projected diagonally. After secondary drying, the dessicator or dessicators containing one batch of material were removed from the refrigerator, and the vacuum released, applying a sterilised pad of cotton-wool between layers of muslin against the nozzle of the dessicator tap.

The laboratory is fitted with Bottogas, and for constricting the ampoule necks the Standard Bottogas blowpipe burner was used, with air from a small rotary compressor (Edwards & Co., type RB4).

With one tray of dessicant in the drying-machine chamber, 90 drawn-out ampoules were loaded on to the three headers, evacuated to 0.3 to 0.2 mm. of mercury for 5 to 10 minutes, and sealed off. A second lot of 90 ampoules was meanwhile drawn out, then loaded, evacuated, sealed, and so on.

For sealing the ampoules no satisfactory Bottogas hand sealer could be found. The Chance's " Flamemaster " torch, with double tip attachment, and added air, was tried and found to be unsatisfactory. A double-flame hand burner requiring no added air was made up by the Bottogas company (after consultation in their workshops) from $\frac{3}{8}$-in. (external) brass tubing, using their No. 1 jets, and with flattened brass flame tips.

Testing and Storage of Sealed Ampoules.

Each sealed ampoule was examined and tested with a high-frequency discharge tester (Edwards & Co., type T2). With freshly sealed ampoules, the

discharge may have to be applied repeatedly for up to 60 seconds at a time before a positive result is obtained. Ampoules which have been stored for a day or longer seldom show this delayed discharge phenomenon.

An immediate loss of 171 ampoules out of a total of 3,021 sealed was experienced.

Sealed ampoules are stored in the deep-freeze cabinet at —20° C.

Testing

For details of reconstitution, safety testing and titration, reference should be made to Priestley (1955*b*). The vaccine reconstituted in agar was inoculated in the tail of cattle, in a dose of 0.2 ml. A minimum number of 10,000 viable organisms has been suggested for field immunisation.

Tube dilution counts (tenfold) in serum broth containing 200 units of penicillin per ml. were carried out on all batches, using the pooled contents of five ampoules from each batch. Growth was obtained up to at least 10-8 dilution in all batches.

Contaminants, mainly large bacilli, and sufficient to show up in stained films which were examined from each of the serum-water suspensions of the organisms harvested from the centrifuge bowl, occurred during Sharples centrifugalisation. Handling of the material during filling out and drying probably gave rise to further contamination. Contaminating organisms grew in some of the tubes of the dilution counts, but were diluted out at worst at a dilution of 10-5.

From Priestley's published results (1955*b*) it would appear that the minimal number of organisms required to immunise when mixed with agar is much smaller than was originally thought. It may, therefore, be possible to freeze dry 0.1 ml. of culture without concentrating it and still have 100 or more doses in the final product. This would obviate the necessity for centrifugalisation and, besides simplifying the procedure, would remove the main cause of contamination. If, however, volumes substantially greater than 0.1 ml. have to be freeze-dried to obtain 100 doses per ampoule, then the advantages of direct drying (without concentration) would largely be lost.

Acknowledgments

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THE TREATMENT OF "PULLET DISEASE" IN FOWLS

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AVIAN monocytosis, fowl nephritis, "visceral gout," renal mononucleosis, or "pullet disease" are only a few of the synonyms for a condition which has become one of the commonest causes of economic loss in laying flocks in this country. This disease was first described by Beaudette in America in 1929 and by Gordon and Blaxland in this country in 1945. Since it was described by the British workers the disease has been diagnosed and confirmed in every part of the country and there can be no doubt that it is now widespread in Britain.

The disease usually occurs in young birds in the early stages of egg production, but growing birds, cockerels and mature two-year-old hens are susceptible. Outbreaks can occur at any time of the year but it is most prevalent during the summer and autumn. An outbreak may be acute, subacute or chronic, and as a rule it is sporadic in character.

Symptoms

The symptoms are not constant although nephritis is always present. The birds are dull and dejected, with marked loss of appetite, frequently the crops are distended with sour food, often the head is "heavy" and cyanosed (hence the synonym of "blue comb") and the vent soiled by diarrhœa. There is a marked fall in egg production and some deaths. "Broodiness" may be very obvious.

Post-mortem appearances

As a rule the birds are in good condition. The kidneys show the characteristic lesions of the disease with the accumulation of masses of urates and the serous membranes show hæmorrhages. The pancreas and liver often show small areas of necrosis. The intestinal tract shows evidence of lack of muscular tone, the muscles are dehydrated and many show whitish "fish-flesh" areas.

Cause

The cause of the disease has not yet been identified. Many explanations have been advanced as to the cause of pullet disease, including various food constituents, such as new wheat, but not confirmed. Recently, however, because of the way the disease is known to spread and also because it is reported to respond to antibiotic medication, evidence has accumulated which indicates that the disease may be of an infective origin.

The experience of one poultry project where several breeding farms are maintained may be quoted as an example. An outbreak of the disease occurred on each of three neighbouring farms and continued to linger on for weeks. Eventually, after every means of treatment had been tried unsuccessfully, the disease died out. When the outbreak appeared to have subsided, a pen of

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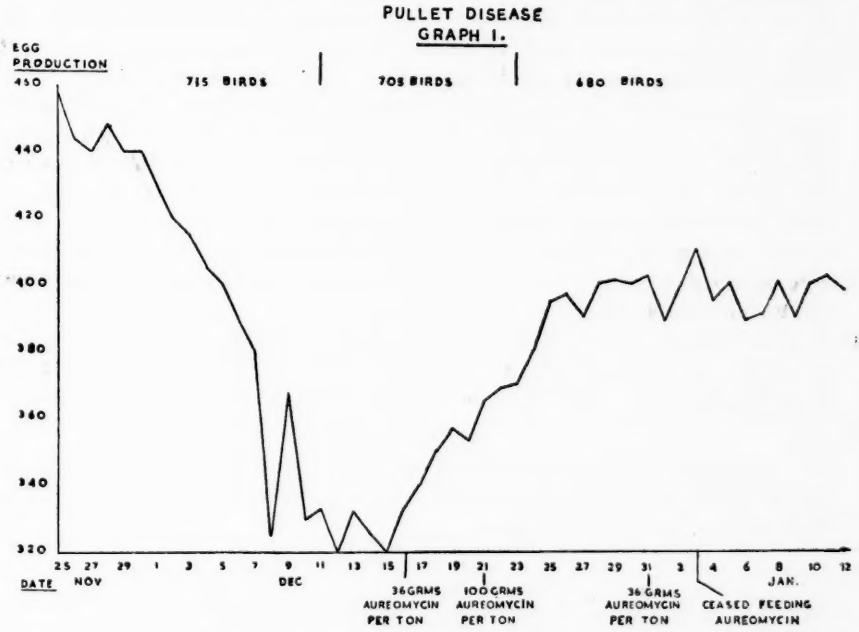
apparently healthy breeding cockerels was taken from one of these farms to another of their breeding farms some miles away, and which had until then been clear of the disease. Within ten days the disease appeared in the indigenous fowls on the farm. This outbreak spread rapidly until a supplement of Aureomycin in the form of Aurofac 2A was given. This provided aureomycin



hydrochloride at the rate of 36 grammes per ton of food. Within ten days the outbreak had subsided and egg production had returned to a satisfactory level.

Recently an outbreak of "pullet disease" occurred in a flock of over 700 Light Sussex pullets on a farm in Berkshire. The egg production was reduced from approximately 460 eggs from 715 birds, or 64.3 per cent production, to 320 eggs or 44.7 per cent after 17 days. At this stage, 10 broody birds were removed for post-mortem examination. For the following four days egg production and ailing birds showed no improvement, when 36 grammes of Aureomycin hydrochloride as Aurofac 2A per ton of food were given and the general demeanour and appetite in particular improved immediately, production rising to 360 eggs per day. At the suggestion of a visitor from America well versed in American methods of treating this disease with Aureomycin, the supplementation was increased to 100 grammes per ton when the production leapt forward to almost 400 eggs per day from only 680 birds, the flock having been relieved of 25 "broody" hens, of which there had been many.

Eventually, the medication was reduced to 36 grammes and then discontinued altogether. The results are shown in Graph 1.

Treatment

The experience gained in this outbreak shows that the decline in egg production may not be obvious for a few days but loss of appetite is immediately very marked. It is not unreasonable to assume that a "shock" treatment given at once, when the loss of appetite is first noted, of 100 grammes of Aureomycin hydrochloride per ton of meal, might check any falling in production and reduce the economic losses from this disease. This treatment should be continued for three or four days only when the quantity of Aureomycin can be reduced to 36 grammes per ton and eventually discontinued two or three days later.

Two other pens of birds similarly affected and left untreated continued to be a source of trouble during the period of recovery in this experimental pen. Since that time opportunity has occurred to treat a considerable number of affected flocks in a similar fashion and reports have been received showing a uniform and encouraging response to the use of Aurofac.

Birds similarly affected, which on post-mortem show excessively flaccid intestines, catarrhal enteritis and atrophy of the gizzard, have responded when treated in this manner.

It is hoped to investigate whether response will follow the feeding of a level of 36 grammes per ton throughout, since in this controlled test, as will be seen from the graph, the response from that level was most encouraging.

Summary

An outbreak of apparent pullet disease is described in a flock of Light Sussex pullets. The pullets responded immediately to supplementation of the diet with high levels of Aureomycin hydrochloride.

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STUDIES ON CONTAGIOUS BOVINE PLEUROPNEUMONIA

With Special Reference to the Complement-Fixation Test

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Part II

(ii) *Serological reactions in naturally-infected animals.* Infected animals can usually be detected easily by means of the C.F. test, whether showing symptoms or not. In acute cases, titres up to 1/2560 have been observed. Of 22 cases in herds under observation, blood-tested on a single occasion only, usually at the time of slaughter, two had titres of 1/40, four 1/160, six 1/320, five 1/640, four 1/1280, and only one 1/2560. Most of these were clinical cases, but one was an apparently healthy animal slaughtered because it was positive to Newing and Field's rapid-slide test, and found to harbour a large and recently encapsulated sequestrum with pleural adhesions. The titre of this

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animal was 1/320. In an acute outbreak in one of the herds of this laboratory where the sera of 50 cases were C.F. tested on a number of occasions (21 fatal cases and 29 sold to the butcher), the maximum titres observed were one 1/10, two 1/20*, four 1/40, six 1/80, four 1/160, twelve 1/320, fifteen 1/640, and six 1/1280. Positive reactions are sometimes noted in animals showing no lesions at post-mortem examination, but the titres are seldom higher than 1/80. Such cases are probably either in the incubation stage, or else recovering and in the process of eliminating the causal organism from the body. Similar cases have been observed in Australia (Campbell and Turner, 1936) and pleuropneumonia organisms have been isolated from lymph glands, etc. In Nigeria attempts at culture from such cases have been unsuccessful. Cases where infection is present but the serum is negative are extremely rare. One definite case is recorded by Campbell and Turner (1953) who suggest—following the observations of Nakumura, Futamura and Watanuki (1926)—that excess antigen in the circulating blood may have removed the antibodies. This was a case with a sequestrum containing viable organisms. One case was observed in this laboratory which was diagnosed macroscopically as pleuropneumonia at post-mortem examination although the serum was negative three days before death, but there is reason to believe that this may have been a mistaken diagnosis and really a pneumonic case of hæmorrhagic septicæmia which is observed sporadically in the area and which can occasionally resemble pleuropneumonia very closely. (A means of differential diagnosis will be discussed later.)

Observations on the laboratory herd confirmed the rapidity with which titres can develop in natural cases and fatal terminations ensue, especially when the animals are in poor condition. One case was observed whose titre rose from a trace to 1/640 in three days. Another rose from negative to 1/80 in two days and died of pleuropneumonia seven days after that. Three other cases died of the disease eight, nine and ten days respectively after being negative. These observations emphasise the limitations of the test as a diagnostic method in a country where communications are slow, and it could easily happen that a field officer might receive a laboratory report that an animal was free from infection after it had actually died of acute pleuropneumonia. On the other hand, the test could be useful in detecting carriers in a herd which had apparently recovered from an outbreak; and in herds close to the laboratory where weekly testing and immediate reporting were possible, the test might serve to eradicate infection before the disease had taken too great a hold. This was actually done in one of the laboratory herds, as will be mentioned in the next section.

(iii) *Serological reactions following vaccination.* At the outset of these experiments it was hoped that a direct relationship might be established between the serological reaction to a vaccine and the degree of immunity conferred so that the reaction could be used as a quick means of comparing the efficacy of various vaccines under trial. However, individual reactions to the same strain were found to be extremely variable. Nevertheless, it did appear that where

* These low titres would presumably have increased had the animals been allowed to live longer. In some cases, also, the last test was a week or two before death.

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moderate numbers of animals were used there was a general pattern of reaction that might be expected in a fair proportion of those vaccinated with a good immunising strain, while a poor strain caused few or no reactions. Previous vaccination or contact with the disease, leading to some degree of immunity or sensitisation to the organism, may be among possible causes for the variation in reactions observed. Another factor probably involved is the age of the animal used. It is generally considered that animals less than two and a half years old, or even under three or four years, are unsatisfactory for experimental work with pleuropneumonia (Mettam, Marshall, personal communications), but often animals of suitable age were not available.

The strain of vaccine used in most of these experiments has been KH3/J, the strongest of three strains obtained from Khartoum many years ago for triple vaccination. Now only this third vaccine (sometimes referred to as 3rd PPV) is used normally in Nigeria for field immunisation. Other strains with which this has been compared include the 2nd PPV strain (KH2/F), strain F 48 obtained from Khartoum in 1950 by the kindness of Major F. W. Priestley, and a locally isolated strain, Jaramai Garmu (or JG). When first obtained, F 48 gave very satisfactory reactions. Fig. 1 shows the reactions of three animals given varying doses of an early subculture of this strain. There appears to be a general tendency for an increased dose of culture to give rise to a higher titre, but this is by no means invariable, and in some experiments there has been an apparent tendency for larger doses to be followed by lower titres.

In October, 1950, three animals were vaccinated subcutaneously with 1 c.c. of KH3/J, five with KH2/F, and three with F 48. This last strain, however, had by then been subcultured for a much longer period than that advised by Priestley, owing to technical difficulties experienced at that time in drying off cultures of this organism. In consequence the strain had undergone considerable modification, and was found to be unsatisfactory as an immunising agent. Titres of 1/320 and 1/40 were obtained in two of the animals receiving KH3/J.* The third did not react. Fig. 2 shows the exact reactions observed when eight weeks later they were placed in close contact with some acute clinical cases. No reactions were observed to vaccination with KH2/F and F 48. Two of those vaccinated with F 48 were exposed four to eight weeks later to contact with infected animals, and both contracted the disease in an acute form. The third, and those which had received KH2/F were all re-vaccinated six weeks later with KH3/J and allowed to mix with the infected herd. None of them contracted the disease, and the only serological reactions were partial fixation in two animals 17 days after vaccination, and a trace reaction in one of the others six and a half weeks later.

In the meanwhile, two cases had occurred in a section of the herd hitherto believed to be free from infection, so 30 animals were vaccinated, using a 2 c.c. dose, 15 with KH3/J and 15 with F 48, whose efficacy had not yet been queried. Four more were vaccinated in the tail with pleural lymph and their reactions will

* These animals were tested every 2 to 3 days. Had they been bled at weekly intervals only, like those in the subsequent experiments, the reaction of the second animal would have been overlooked.

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be referred to in the next section. The remaining 32 were left unvaccinated, as susceptible animals were required for experimental use, and an attempt was made to control the spread by weekly testing and prompt removal of reactors before they had become capable of infecting others. Only one of these developed the disease. Two others developed " transient " reactions. These were isolated, and slaughtered after one and four weeks respectively, by which time they had become negative to the test. At post-mortem examination there was no trace of lesions past or present.

The reactions to KH3/J were very much more marked than those to F 48, and are summarised in Table I.

TABLE I.

Maximum titre of reaction.	Number of animals. KH3/J.	F 48.
1/80	1	0
1/40	3	0
1/20	2	1
1/10	1	0
Partial.	3	2
Trace.	2	2
Nil.	2	9
	14	14

One animal in each vaccine group became infected with the disease shortly after vaccination. That given F 48 developed a titre which rose in four weeks to 1/160. It then became a clinical case and started to cough, so it was removed and slaughtered. Post-mortem examination showed it to be a typical acute case. That given KH3/J rose to 1/40 after two weeks and this was at first thought to be a normal reaction. After the fifth week the titre rose to 1/320, and although the animal was apparently healthy and in good condition, it was isolated, and slaughtered two weeks later. One small encapsulated lesion an inch and a half in diameter was found. It would appear that these two animals had either been vaccinated during the incubation period or had become infected shortly after vaccination. F 48 had no apparent effect on the course of the natural disease, but in that receiving KH3/J the disease was arrested with early encapsulation of a single small lesion.

In the majority of the animals reacting to the vaccine the maximum titre was reached at about the 14th day, and only in two was there any appreciable reaction by the seventh. These were isolated and later slaughtered, in case this should have been the beginning of an attack of the disease and not a reaction to the vaccine. However, no trace of lesions past or present could be found post-mortem.

Another point of interest noted in two of these vaccinated animals, one with KH3/J and the other with virulent material into the tail, was that transient low-titre reactions, partial or 1/10, reappeared three months or more after the initial reaction, if any, had disappeared. Nothing similar was seen in any of the

unvaccinated animals. It will be suggested later that such transient reactions occurring in the absence of an infected environment may be taken as indications of some degree of immunity.

The next comparative test with vaccine was 18 months later when, as a pilot experiment to a larger one, three animals received 1 c.c. of KH3/J, three 2 c.c. of the same, three 1 c.c. of JG (43rd. generation), and one 2 c.c. One of those receiving 2 c.c. of KH3/J developed a titre of 1/160 and the other two

TABLE II.

Maximum titre of reaction.	Number of animals	
	Dose 1cc.	Dose 2cc.
1/40	2	0
1/20	14	2
1/10	5	2
Partial.	3	1
Trace.	6	8
Nil.	1	3
	31	16

1/20. Of those given 1 c.c. one developed a titre of 1/10 and the others only partial or trace reactions. Details are shown in Fig. 3. (That with 1/160 is omitted as it died from other causes before the challenge half of the experiment could be commenced.) That given 2 c.c. JG did not react at all, and those receiving 1 c.c. only showed slight traces. Accordingly KH3/J alone was used in the main experiment, and when the pilot animals were challenged the uselessness of strain JG was confirmed.

In the larger experiment 47 animals were vaccinated with varying doses of KH3/J. Unexpectedly, better reactions followed the 1 c.c. does than the 2 c.c. (Table II.)

This was tentatively attributed to the fact that the animals in question were a very mixed batch as regards age, and were of unknown history. A repeat test was therefore made on two small batches of animals of known history, never vaccinated or in contact with the disease, and of uniform ages. (Table III.)

Insofar as so small a group of animals can show anything, this test would add support to the view that in spite of the results in the previous experiment, the general tendency among older animals, e.g., two and a half years old upwards, is for a larger dose to produce a higher titre. In younger animals, results are more erratic.

(iv) *Serological reactions following the use of virulent material.* At the time when half of one of the laboratory herds was vaccinated, as mentioned in the previous section, four animals were vaccinated into the tail with 0.5 c.c. of pleural exudate collected 24 hours previously and preserved with 25 per cent glycerine to control contaminants. No visible reactions developed. One developed a titre of 1/160, two were 1/20, and the fourth showed only partial fixation. This last animal, challenged after ten months, was found to be solidly immune. Those

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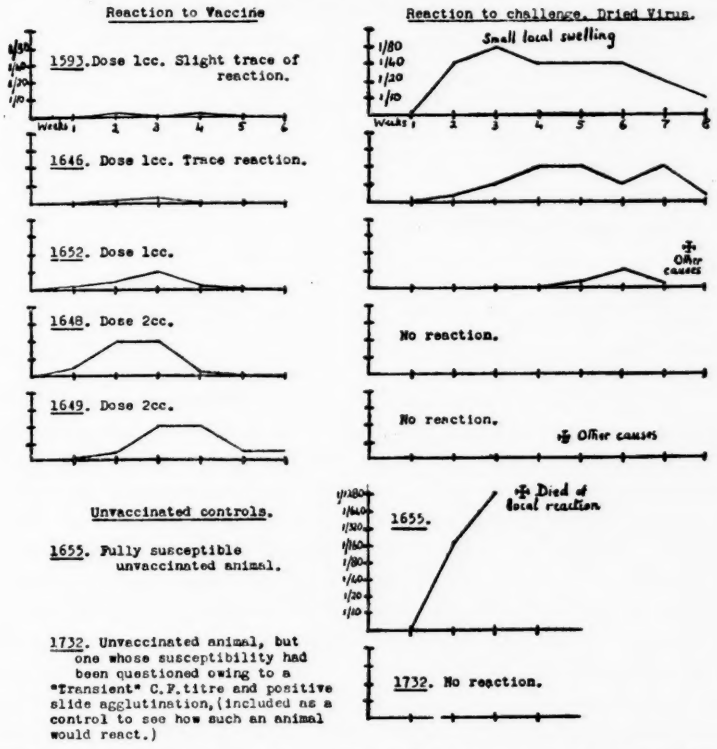
with titres of 1/20 both showed transient reactions (1/10). one eight weeks and the other 16 weeks after inoculation. The latter animal was slaughtered but no abnormality was found post-mortem. Neither animal was in an infected environment at the time. At the same time as these tail vaccinations two other animals were given the same inoculum subcutaneously over the nasal bones in order to see the type of reaction resulting from this method. One developed a large swelling, commencing about the 10th to 12th day, and causing death after

Fig. 3. Reactions to vaccination (KH3/J) and subsequent challenge.


Reaction to Vaccine
Reaction to challenge. Dried Virus.
1593. Dose 1cc. Slight trace of reaction.
Small local swelling
1646. Dose 1cc. Trace reaction.
1652. Dose 1cc.
Other causes
1648. Dose 2cc.
No reaction.
1649. Dose 2cc.
No reaction.
Other causes
Unvaccinated controls.
1655. Fully susceptible unvaccinated animal.
Died of local reaction
1655.
1732. Unvaccinated animal, but one whose susceptibility had been questioned owing to a "Transient" C.F. titre and positive slide agglutination, (included as a control to see how such an animal would react.)
1732. No reaction.

four weeks. The titre was 1/160. The other showed no local reaction, but died about the same time. Death was attributed to parasitic infection. The C.F. titre was partial only.

Six animals were inoculated subcutaneously behind the shoulder with 1 c.c. of freshly collected 20 per cent glycerinised pleural exudate, as controls in an experiment where the same exudate was being used after treatment with chemicals. It is at this site that reactions are likely to be most severe. Four of them developed large swellings with extreme lameness and had to be slaughtered four to five weeks after inoculation. At this time they were unable to stand, and the maximum titres varied from 1/160 to 1/2560. A fifth developed a much smaller swelling and a mild transient lameness, but the titre rose to

1/1280. It remained in good health all the while and the titre had fallen to 1/80 by the end of the 12th week. It remained steadily at this level, with only minor fluctuations, for more than a year, when it was finally slaughtered. This case (No. 1316) will be discussed in more detail later. The remaining animal only developed a very small swelling and was never lame. The serological reaction was slower than the others, and at the end of four weeks (when most of the rest were being slaughtered, it was still only 1/20. However, by the fifth week it had risen to 1/320 and remained at this level until it died during the seventh week. In spite of the smallness of the swelling the post-morten

TABLE III.

2½ year old animals.			15 month old animals.		
Maximum titre.	1cc	2cc.	Maximum titre.	1cc.	2cc.
1/80	0	1	1/20	1	0
1/10	0	1	1/10	1	1
Partial.	0	1	Partial.	0	0
Trace.	3	1	Trace.	1	1
Nil.	3	2	Nil.	0	1
	6	6		3	3

TABLE IV.

Animal number-	Titres at the end of :- 1 week.	2 wks.	3 wks.	4 wks.	5wks.	Remarks.
1289	Nil	Trace	1/160	1/320	1/2560	Slaughtered. Large swelling & severe lameness. Unable to stand.
1338	Nil	1/20	1/320	1/320	-	
1370	Trace	1/40	1/320	1/80	-	
1381	Nil	Nil	1/160	1/160	-	
1362	Nil	Nil	Trace	1/20	1/320	Small but typical swelling. Not lame. Died after 6 wks.
1316	Nil	1/40	1/640	1/640	1/1280	Small swelling, transient lameness. Titre 1/80 by 12th week & remained same for 15 months.

appearance of the subcutaneous tissues was that of a typical reaction, although the latter was not extensive. It is probable that the local lesion was not the sole cause of death which may have been accelerated by parasitic infection. Details of the reactions are given in Table IV.

In subsequent experiments animals were inoculated with dried pleural exudates, as natural cases of the disease were no longer available for the supply of fresh material. The results obtained in 22 animals, controls in challenge experiments or inoculated in order to obtain fresh lymph, were substantially the same as those with the fresh exudates. With the fresh exudate there were five fatal reactions out of six, and with the dried, 20 out of 22. The maximum titre was most commonly 1/320, although up to 1/2560 were observed. Usually the peak was reached during the third week and deaths occurred most frequently during the fourth. As a rule serological reactions did not start until the second week. Only one animal in this series had developed a titre (1/10) by the seventh day.

(v) *Serological reactions in other species of animal.*---Two rabbits were inoculated subcutaneously with an emulsion of diseased lung tissue from an apparent case of pleuropneumonia to see whether any *Pasteurella* could be isolated. Both survived and no pathogens were recovered. Their sera were meanwhile being C.F. tested for pleuropneumonia antibody, and showed rapidly developing high titres. One reached its maximum (1/640) on the eighth day, and the other (1/320) on the 11th. The reaction persisted until the 46th day.

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Six more rabbits were then inoculated with reconstituted dried pleural exudate in doses varying from 1 c.c. to 0.01 c.c. to see what was the smallest dose required to produce a definite serological reaction. None of them reacted. In case this should have been due to any loss of virulence in the dried material, which was now over a year old, a further ampoule of the same batch was used to inoculate two cattle. Both reacted typically, with no reaction by the seventh day but thereafter rising to a peak of 1/1280 on the 18th day in one, and 1/640 on the 23rd in the other. "Tumour-lymph" (fluid oozing from the cut surface of the local swelling) was collected from one of these at post-mortem and immediately inoculated into two more rabbits. On the seventh day these had titres of 1/640 and 1/160. They died from abscess formation at the site of inoculation on the ninth and tenth days respectively, before further sera had been collected. On a later occasion four rabbits were inoculated with tumour lymph, two fresh rabbits and two of those which had been given dried pleural exudate nine months previously without reacting. The two former reached titres of 1/320 and 1/160 on the seventh day. One of those previously inoculated was 1/40 on the seventh day (dying of pyæmia shortly afterwards) and the other was 1/10, rising to 1/40 on the 13th and then falling.

This observation suggests a useful means of confirming cases of pleuropneumonia found at post-mortem examination when serum samples are not available. Rabbit inoculation has since been used successfully where an acute pneumonia was present. Probably it is not suitable for small sequestra or resolving lesions, where organisms may be present in too small numbers to evoke a serological response. The rabbits frequently die of local abscess formation, but as they usually live for over a week, and the serological reaction has in most cases started by the fourth day and is quite high by the seventh, this is of no consequence. Were an acute pasteurellosis the cause of the pneumonia, this would reveal itself by the rabbit's dying of the typical septicæmia. The rabbit test is, therefore, a useful means of differential diagnosis between pleuropneumonia and cases of hæmorrhagic septicæmia of the type which resembles the former disease so closely.

A similar experiment has been made with sheep. Tumour lymph was collected from an ox reacting typically to subcutaneous inoculation of dried pleural exudate and inoculated subcutaneously into two sheep. Two other sheep received a similar dose of freshly reconstituted dried exudate from the same batch as that used to obtain the bovine tumour lymph. The sheep receiving the dried exudate at no time showed more than a trace of fixation. The sheep receiving the tumour lymph had developed titres of 1/40 and 1/20 by the fifth day, and by the 12th these were 1/320 and 1/40 respectively, after which they began to fall. One of these sheep developed a small swelling at the site of inoculation. As it survived, the pathological anatomy of the swelling was not studied, and it cannot be said whether it was a typical reaction to the pleuropneumonia organism comparable with that seen in cattle, or whether it was merely a reaction to some other contaminating organism present in the lymph. Campbell (1936) refers to subcutaneous reactions in sheep inoculated with the bovine organism,

but gives no details of the type of lesion. Beller and Tahssin-Bey (1926) claim to have infected sheep, and Dujardin-Beaumetz (1906) produced a lesion comparable with that seen in cattle given virulent material subcutaneously, but only by injecting doses of 50 to 100 c.c. of culture.

(Part III will appear in March issue)

ARTHROPLASTIC TREATMENT FOR OSTEOCHONDRITIS COXA IN THE DOG

By CHURCHILL FROST and G. C. KNIGHT
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THERE is evidence that many dogs suffering from severe lameness due to osteochondritic lesions of the femoral head, regain useful function of the limb spontaneously in about four to six months from the onset of the symptom. When the lameness persists for a much longer period, prognosis is unfavourable and permanent disability, at least, in the use of the leg can be expected. In some such cases during the past few years, the possibility of practical arthroplastic surgery has been considered and one, so far, satisfactory replacement of the femur head in a case of osteochondritis coxa is recorded.

The subject, a miniature poodle dog aged 18 months, with a history of lameness on the right hind leg for nine months, was examined and found to have a comparatively prominent great trochanter, no marked discomfort on manipulation of the hip joint and evident shortness in the length of the leg, all these signs being characteristic of osteochondritis coxa. X-ray examination showed resorption and deformity of the femoral head and hyperthrophy of the acetabular margin (Illustration No. 1). In the circumstances prognosis was thought to be unfavourable but it was arranged there should be re-examination in a month. At this second examination no improvement in the function of the leg could be detected and it was decided to design a femoral head prosthesis. Its preparation was expected to take a few weeks and the replacement operation was planned to take place as soon as the prosthesis became available, providing the lameness had not improved by that time.

From the X-ray appearances of the normal left femoral head, a drawing was made of a ball head prosthesis having an hexagonal base with a central tapping for a securing stem screw. This screw was to be of sufficient length to transfix the femur from a point below the great trochanter at an angle of about 45° with the long axis of the bone and so maintain the prosthesis in the appropriate position. The screw and prosthesis were to be of non-toxic stainless steel and a set spanner provided to fit the hexagonal base in order to prevent rotation while the screw was tightened. Without damaging the polished articular surface, no simple means of holding the prosthesis could be conceived other than by the provision of some non-articular area for the purpose (Illustration No. 2).

The third examination of the dog was made about one month after the second examination and there was no appreciable improvement in the use of

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(*Article by Frost and Knight, page 86*)

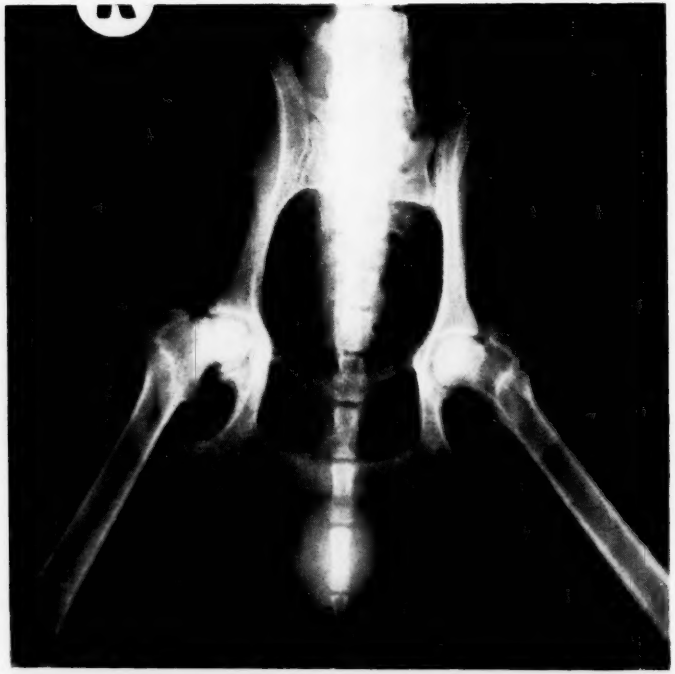
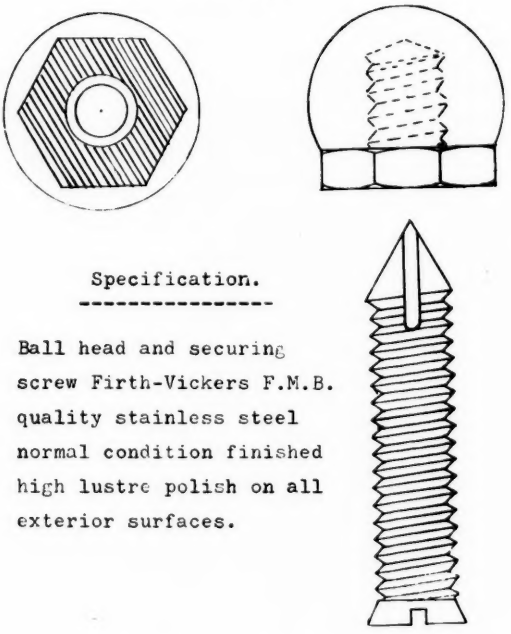


Illustration No. 1.

Illustration No. 3.

Scale 1:3.



Specification.

Ball head and securing screw Firth-Vickers F.M.B. quality stainless steel normal condition finished high lustre polish on all exterior surfaces.

Illustration No. 2.

(*Article by Frost and Knight, page 86*)

the right hind leg. By this time the prosthesis had been received and an operation was arranged.

The Operation.

The limb was prepared by clipping hair from well above the hip to the hock and followed by shaving a wide area down to the mid-thigh level. Chlorpromazine and atropine premedication preceded thiopentone sodium given for induction, anæsthesia then being maintained by nitrous oxide, trichlorethylene and oxygen. For postural hypotension, hexamethonium bromide was used.

A skin and subcutaneous tissue incision made from a point about 5 cm. above the great trochanter and continued down to the mid-thigh level exposed the superficial, the middle gluteus and tensor fascia lata muscles. The hip joint was approached by amputation of the great trochanter at a point just below the attachment of the superficial, middle gluteus and piriformis muscles. These muscles were reflected back with the amputated trochanter.

By incision of the deep gluteus muscle and of the underlying joint capsule, the articular head of the femur was exposed sufficiently to allow section of the teres ligament with scissors. This freed the femoral head to an extent facilitating its amputation in the plane of the metaphysis.

Preparation for mounting the prosthesis was then made, first by incising down the tensor fascia lata and lateral vastus muscles for about 2 cm. from the level at which the trochanter had been amputated, and then by drilling the bone via this incision through the cortex to the centre of the metaphyseal plane. Having passed the stem screw through the hole drilled for it, the prosthesis was mounted, held with the spanner to prevent rotation and drawn firmly down to the metaphysis by tightening the screw.

The replacement femoral head was then articulated with the acetabulum. As far as possible, the joint capsule was restored into place and the deep gluteus incision closed with three sutures of 20 day 3/0 chromic catgut. Having drilled horozontally through the amputated trochanter and similarly through the femoral base, a strand of 30 S.W.G. braided stainless steel was passed through these holes, tied and the trochanter so held in the normal position. Replacing the trochanter had the effect of preventing luxation of the femoral head in spite of no contribution being made by the capsular and teres ligaments. Interrupted 3/0 chromic catgut sutures were used to close incisions in the tensor fascia and lateral vastus muscles and to take up subcutaneous tissue dead space before finally suturing the skin wound with nylon monofilament over a gauze pad. The operation time was about two hours.

The dog, having made a normal recovery from anæsthesia which had been maintained for a period of two hours ten minutes, was discharged into the care of the owner the following day. On each of the two days before operation and on each of five days after, 300,000 units penicillin was given intramuscularly.

On the eighth day following operation, the skin sutures were removed and at that stage a little weight was occasionally being taken on the limb. No further examination was made for another three weeks when the limb function was

quite normal and manipulation apparently causing no discomfort (Illustration No. 3). Another examination 5 weeks after the operation showed the condition to be unchanged. Although the evidence and experience available up to the present is quite insufficient for any long term prediction. the findings at regular future examinations are awaited with interest.

Thankful acknowledgment is made to Miss B. M. Q. Weaver for her skilful management of the anæsthesia and to Miss E. M. Harrod, Mr. H. A. Burgess and other colleagues for their help. Messrs. Arnold & Sons, of Wigmore Street, London, W.1, made the prosthesis. To all concerned in this we are grateful for the interest shown.

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REVIEW

DISEASES OF THE PIG, by David J. Anthony, M.R.C.V.S., D.V.S.M.(Vict.), F.R.San.I. Fourth Edition. London: Baillière, Tindall & Cox. Pp. xii + 370 with 81 Illustrations. Price 25s.

THIS fourth revised and enlarged edition provides a valuable contribution to the literature on Pig Diseases. The general format of the book remains the same as in previous editions; preliminary chapters deal with the Breeds of Pigs, their Housing, Management and Feeding, and the main substance of the book covers their diseases comprehensively and with commendable emphasis on the practical aspects of the subject. There is considerable amount of new material. The new section on Virus Pneumonia of Pigs being of particular value. The photographs accompanying the text are excellent. The book is an improvement on the previous edition which was acknowledged as a standard reference book on Pig Diseases.

A word of praise must be reserved for the publishers, Baillière, Tindall & Cox, Ltd. since the book is not only very reasonably priced, but the standard of the printing, paper and binding leaves nothing to be desired.

ERRATUM

In the January issue, in an article by J. B. Tutt, M.R.C.V.S., the word "Streptoquaine" was mis-spelt and should have read as "Streptaquaine." This is a product of Distillers Co. (Biochemicals) Ltd.

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